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THE PHILIPPINE
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AND THE INDUSTRIES

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THE SOLUBILITY OF PORTLAND CEMENT AND ITS RELATION TO THEORIES OF HYDRATION [1]

By J. C. WITT and F. D. REYES

(From the Laboratory of General, Inorganic, and Physical Chemistry, Bureau of Science, Manila, P. I.)

ONE TEXT FIGURE

In connection with some previous work in this laboratory,[2] occasion arose to treat a few grams of cement with a solution of sodium sulphide and then to filter, wash, and examine the filtrate. It was found impossible to wash the residue free from soluble calcium compounds, for the wash water invariably showed a test for that element. Moreover only a portion of the calcium compounds dissolved came through the filter, because calcium carbonate was formed from contact with the air. The same behavior was noted when water was substituted for the sulphide solution. It was found that many times the original quantity of water could be added without resulting in a residue free from soluble calcium compounds. This suggested an investigation to determine just what constituents of cement will go into solution and the proportion of the total amount of each present in the sample. A review of the literature revealed that, while a number of writers mentioned the solubility of constituents in water, there were few reliable quantitative data available.[3]

It is common experience that water in which cement test pieces are stored soon contains substances in solution. It becomes soapy to the touch and has an alkaline reaction, and a qualitative test will reveal the presence of the calcium ion. That calcium hydroxide is among the products of the hydration of cement

[1] Received for publication April 10, 1918.

[2] Witt, J. C., *This Journal, Sec. A* (1916), 11, 273.

[3] Compare, however, Winkler, A., *Journ. prakt. Chem.* (1856), 67, 444.

has been well established. Le Chatelier [4] observed crystals of calcium hydroxide in examining sections cut from hardened cement specimens. Winkler [5] says that cement is hydrolyzed into free lime and some compounds of lime, silica, and alumina. Stern [6] found that calcium aluminates were decomposed by water, forming gelatinous alumina and calcium hydroxide. Reed [7] made some interesting microscopic studies of hydrated cement. He says:

> When Portland cement is gauged with water, lime goes into solution and a thin skin of calcium carbonate is formed on the moist surface which protects the interior mass more or less completely from the action of the air.

His method is to treat cement with water on a microscope slide and then to protect the mixture from the air by paraffin. Two kinds of crystals are formed—calcium aluminate and calcium sulphaluminate. Hart [8] mixed cement with water and then filtered rapidly. The filtrate contained sulphates, silicates, free lime, and caustic alkali. On letting a fresh mixture stand two and one-half hours and then filtering and examining the filtrate, he found the chief constituent to be potassium sulphate, which he concluded was formed by the double decomposition of the soluble potassium compounds and the calcium sulphate present.

PRELIMINARY WORK

The term solubility as employed in this paper signifies the mass dissolved per gram of cement present in the system under given conditions and not the mass dissolved by a unit weight of water—as in most solubility measurements. The proportion of water has been kept considerably in excess of the amount necessary, and no effort was made to keep the temperature constant. All the experiments were made at room temperature in Manila, which averages about 28° to 30° C. The factors that influence the results have been found to be fineness of grain, quantity of water present, absence of carbon dioxide, method of agitation, and time.

Four brands of cement, which we shall designate as I, II, III, and IV. were used in the work. The analyses are shown in Table I.

[4] Cf. West, C. H., The Chemistry and Testing of Cement. London, Edward Arnold (1911), 113.

[5] Loc. cit.

[6] Stern, E., *Chem. Zeitg.* (1908), 32, 1029.

[7] Reed, E. J., *Journ. Soc. Chem. Ind.* (1910), 29, 735.

[8] Hart, *Tonind. Zeitg.* (1908), 32, 754. [*Journ. Soc. Chem. Ind.* (1908), 27, 568.]

TABLE I.—*Analyses of cements.*

[Numbers indicate percentages.]

	Brand.			
	I.	II.	III.	IV.
Loss on ignition	2.43	2.15	2.17	3.24
Silica (SiO_2)	22.60	21.40	21.26	20.62
Alumina (Al_2O_3)	7.72	7.58	8.54	6.62
Ferric oxide (Fe_2O_3)	1.76	1.70	2.08	2.56
Calcium oxide (CaO)	61.32	62.94	62.82	63.50
Magnesia (MgO)	1.08	1.37	1.13	1.43
Sulphuric anhydride (SO_3)	1.45	1.61	1.02	0.82
Sodium and potassium oxides (Na_2O, K_2O)	1.63	1.14	1.17	1.33

Five hundred cubic centimeters of water were placed in each of four 800 cubic centimeter Erlenmeyer flasks, fitted with a rubber stopper with two holes. Through one of these holes was inserted a reflux condenser; the other carried a glass tube bent at right angles, the end of which projected beneath the surface of the water. The water was boiled for an hour or two by means of a Bunsen burner, until all the dissolved gases were expelled. The flame was then removed, and a current of air free from carbon dioxide [9] was passed through the tube into the liquid, until the flask and contents had assumed room temperature. In the meantime two grams of cement were weighed into a small glass bulb. While the current of air was still passing, this bulb was dropped into a flask. The two-hole stopper was replaced with a solid stopper and the contents vigorously shaken to prevent the cement from caking. The flask was then placed in a mechanical shaker and vigorously agitated for twenty hours. Of the several types of shakers available for this work, the most satisfactory may be described as follows: A small platform was mounted on grooved wheels, which were supported by a small track. This platform was rapidly driven back and forth by a crank shaft, having a stroke of about 12 centimeters. The flasks were clamped in a horizontal, longitudinal position. The flask was then removed and allowed to stand for twenty-four hours, when the solid matter completely settled, leaving a clear supernatant liquid. The stopper was partly removed, and the tube was inserted in the neck of the flask, through which a

[9] The air was passed through two wash bottles containing potassium hydroxide solution and then through one containing barium hydroxide solution. The last named acted as an indicator. If a trace of carbon dioxide escaped from the first two bottles, it was caught in the third and produced a turbidity. The contents of all of the bottles were then changed.

current of air free from carbon dioxide was passed. A pipette was then inserted, and portions were withdrawn for analyses.

The solids in the bottom of each flask consisted of two layers. The upper was white and flocculent, consisting partly of aluminium hydroxide. The lower was much larger and evidently consisted partly of cement which had not been decomposed by the action of the water. Analysis of the supernatant liquid showed it contained in solution considerable calcium, a trace of iron and aluminium, and no silica. The shaking was repeated for another period to see if any more calcium went into solution. Analysis showed there was an appreciable increase. This was repeated until the flasks had been shaken for a total of four hundred twenty-six hours. Numbers II and IV had become constant, and number I practically so, but number III still showed a gain. The lower layer of solid in the flask had almost entirely disappeared, only a few particles remaining. During the experiment it was necessary to add more water from time to time to keep the solution from reaching saturation. It was decided to stop the work at this point and start a new series after making a number of changes that the work had suggested. The results of the first series of tests are given in Table II. It will be noted that from 35 to 38 per cent of the total calcium of each cement went into solution.

TABLE II.—*First series. Calcium dissolved from original cements during various periods of shaking with carbon dioxide free water.*

[Numbers indicate weight of calcium, in grams, per gram of cement.]

	Brand.			
	I.	II.	III.	IV.
Total calcium (Ca) in cement	0.4383	0.4499	0.4490	0.4539
Calcium (Ca) dissolved during each period:				
First period, 20 hours	0.0665	0.0808	0.0843	0.0681
Second period, 16 hours	0.0071	0.0125	0.0089	0.0259
Third period, 20 hours	0.0129	0.0245	0.0172	
Fourth period, 40 hours	0.0086	0.0114	0.0115	[a] 0.0288
Fifth period, 55 hours	0.0168	0.0130	0.0114	0.0129
Sixth period, 48 hours	0.0255	0.0208	0.0138	0.0184
Seventh period, 22 hours	0.0071	0.0038	0.0063	0.0053
Eighth period, 41 hours	0.0106	0.0066	0.0052	0.0000
Ninth period, 54 hours	0.0092	0.0007	0.0010	
Tenth period, 45 hours	0.0041	0.0000	0.0029	
Eleventh period, 65 hours	0.0020		0.0104	
Total, 426 hours	0.1644	0.1741	0.1729	0.1594
Total calcium dissolved (per cent)	37.51	38.69	38.50	35.12

[a] This value represents the amount dissolved during both third and fourth periods.

MANIPULATION

Since it was likely that the larger particles of cement were the last to be affected by the water, these were eliminated before starting the second series. An air separator essentially similar to the Goreham flourometer [10] was utilized. No attempt was made to obtain quantitative results nor to measure the size of grain. The air pressure corresponded to 20 millimeters of mercury. The air was passed through suitable solutions to remove both moisture and carbon dioxide, before coming into contact with the cement. Since cement dust is likely to be slightly different in chemical composition from the original cement after such a separation, the cements were again analyzed, with the results shown in Table III. All the work hereafter described was done with this material.

TABLE III.—*Analyses of cements after air separation.*

[Numbers indicate percentages.]

	Brand.			
	I.	II.	III.	IV.
Loss on ignition	3.91	3.73	3.55	5.00
Silica (SiO_2)	20.48	20.22	18.96	18.40
Alumina (Al_2O_3)	7.81	7.17	9.58	8.95
Iron oxide (Fe_2O_3)	2.37	2.11	2.32	2.03
Calcium oxide (CaO)	61.14	62.38	61.84	62.20
Magnesia (MgO)	1.22	1.30	1.42	1.34
Sodium and potassium oxides (Na_2O, K_2O)	1.07	0.64	0.63	0.87
Sulphuric anhydride (SO_3)	1.97	2.43	1.62	1.27

Some other changes also were found advisable before starting the next series of determinations. It was found that the Erlenmeyer flasks did not stand the continued rough usage in the shaking machines. It was also desirable to increase the actual amount of water for each experiment as well as the quantity per gram of cement. Therefore the new manipulation was as follows:

A 20-liter bottle was filled with water free from carbon dioxide and protected by a soda-lime bulb. A special automatic pipette was made with an approximate capacity of 850 cubic centimeters. When this was standardized, it was found to deliver 863.5 cubic centimeters. This value was constant and was sufficiently close to the desired volume, so it was not changed. The pipette was mounted and then connected with a siphon in the 20-liter bottle.

[10] Cf. *Tech. Paper, U. S. Bur. Standards* (1915), No. 48, 8.

The air inlet was protected by a soda-lime bulb, so that the water could be easily and quickly measured and delivered without exposure to carbon dioxide. Narrow-mouthed glass-stoppered bottles were substituted for the Erlenmeyers.

To start one of the new series of experiments, it was only necessary to wash out a bottle with air free from carbon dioxide, place therein a pipetteful of water, and quickly add a glass capsule of cement previously weighed. Only a trace of carbon dioxide was present in the system. For each gram of cement, 431.75 cubic centimeters of water were present. At 30° C. 400 grams of water are sufficient to dissolve 0.612 gram calcium hydroxide,[11] which is equivalent to 0.462 gram calcium oxide, or 74.06 per cent of the total calcium oxide in the cement containing the most calcium oxide. As will be shown later, the highest percentage of calcium going into solution in this series was 40.89 per cent.

Table IV shows the calcium in solution for each sample of fine cement during fifteen days' shaking, or until each sample had reached a constant value.

TABLE IV.—*Second series. Calcium dissolved from fine cement by shaking with carbon dioxide free water.*[a]

	Brand.			
	I.	II.	III.	IV.
	g.	*g.*	*g.*	*g.*
Total calcium (Ca) present per gram of cement	0.4369	0.4158	0.4119	0.4445
Calcium (Ca) dissolved per gram of cement:				
First period, 1 day	0.1282	0.1432	0.1519	0.1535
Second period, 1 day	0.0072	0.0147	0.0091	0.0113
Third period, 2 days	0.0073	0.0131	0.0076	0.0025
Fourth period, 2 days	0.0034	0.0056	0.0018	0.0038
Fifth period, 2 days	0.0058	0.0013	0.0014	0.0017
Sixth period, 2 days	0.0104	0.0026	0.0021	0.0031
Seventh period, 5 days	0.0041	0.0000	0.0000	0.0000
Total, 15 days	0.1664	0.1805	0.1749	0.1759
Percentage of total calcium that goes into solution	38.09	40.49	39.58	39.57

[a] At the end of the sixth period three of the cements showed constant results. At the end of the seventh period the other one was constant. The total $Ca(OH)_2$ in solution at the end of the operation was well below the saturation point, showing that the constant value was not due to a saturated solution.

The main difference between this series and the first is the much greater amount of calcium going into solution during the

[11] Seidel, Atherton, Solubilities of Inorganic and Organic Substances. D. van Nostrand Co., New York (1907), 99.

first period—about double. The time necessary for completion was shorter, and the percentage of the total calcium present was higher.

The total amounts of other elements in solution are negligible in comparison with the calcium. The complete analysis of the liquid after twenty-four hours of shaking is shown in Table V. The first column under each number shows the amount of each constituent dissolved per gram of cement. The second column shows the percentage of the total amount of each constituent in solution (compare with Table III).

TABLE V.—*The weight of each constituent (per gram of cement) that goes into solution during the first twenty-four hours. Also the percentage of the total amount of each constituent present that is dissolved.*

	Brand.							
	I.		II.		III.		IV.	
	g.	*P. ct.*	*g.*	*P. ct.*	*g.*	*P. ct.*	*g.*	*P. ct.*
Silica (SiO_2)	trace		trace		trace		trace	
Iron and aluminium oxides (Fe_2O_3, Al_2O_3)	0.0088	8.64	0.0065	7.00	0.0037	3.11	0.0064	5.82
Calcium oxide (CaO)	0.1797	29.39	0.1998	32.03	0.2135	34.51	0.2147	34.62
Magnesium oxide (MgO)	trace		trace		trace		trace	
Sodium and potassium oxides (Na_2O, K_2O)	0.0045	42.06	0.0041	64.06	0.0044	69.84	0.0057	65.52
Sulphuric anhydride (SO_3)	0.0131	66.50	0.0183	75.31	0.0111	68.52	0.0096	75.59

There is no important increase in the amounts of these constituents in solution after the first period of shaking. The percentage of each constituent in solution is interesting. We should not expect to find any soluble silicates under these conditions, and the small amount of iron, aluminium and magnesium in solution is not surprising when such a large concentration of calcium hydroxide is present. The absence of magnesium may be partly due to the slowness with which magnesium compounds hydrate. The slight solubility of calcium sulphaluminate explains the small amount found in solution.

After the series of experiments had reached completion, and no further calcium went into solution, the precipitate remaining in each flask was collected and washed. (It was not possible to wash completely free from soluble calcium compounds.) The residues were then analyzed as a check on the analyses of the soluble portion. In Table VI the first line shows the loss on ignition, after the material had been dried to constant weight at 110° C. The other results were calculated to the after-ignition basis, in order that they might be more comparable with the cement before water was added.

TABLE VI.—*Analyses of residue after completion of solubility determinations.*

[Numbers indicate percentages.]

	Brand.			
	I.	II.	III.	IV.
Loss on ignition	25.52	28.64	25.26	26.24
Silica (SiO_2)	35.56	34.57	32.36	33.10
Alumina (Al_2O_3)	14.26	13.40	10.30	10.12
Iron oxide (Fe_2O_3)	2.36	3.17	5.16	4.80
Calcium oxide (CaO)	45.12	45.45	49.00	48.90
Magnesium oxide (MgO)	1.86	2.65	2.20	2.27
Sulphuric anhydride (SO_3)	0.35	0.18	0.53	0.25
Sodium and potassium oxides (Na_2O, K_2O)	0.44	0.50	0.32	0.54

Comparing this with Table III it may be noted that the removal of calcium by solution has considerably raised the percentage of aluminium, silica, and magnesium in the residue. The percentage of iron remains about the same, but the percentage of sulphuric anhydride and alkalies is less because of the high percentage of each of these going into solution.

The next point investigated was the effect of a larger volume of water per gram of cement. Would this affect speed of solution or the final quantity of constituents in solution? The same volume of water was taken in each case, but less cement—2, 1, 0.5, 0.25, 0.1 gram, respectively. The volume of water present per gram of cement was then calculated and, neglecting the decimals, the values are shown in Table VII.

TABLE VII.—*Effect of the volume of water on velocity of solution and total amount of calcium dissolved in twenty-four hours.*[a]

Volume of water per gram of cement.	I.		II.		III.		IV.		Time required for constant results.
c. c.	*g.*	*Per cent.*	*g.*	*Per cent.*	*g.*	*Per cent.*	*g.*	*Per cent.*	*Days.*
432	0.1282	29.34	0.1432	32.12	0.1519	34.37	0.1535	34.52	12
864	0.1537	35.14	0.1857	41.63	0.1816	41.07	0.1816	40.84	12
1,727	0.1993	45.60	0.2407	53.96	0.2444	55.27	0.2334	52.48	1
3,454	0.2707	61.92	0.2934	65.79	0.2934	66.36	0.2690	60.48	1
8,635	0.4038	92.41	0.3907	87.63	0.4038	91.36	0.3855	86.65	1

[a] The first column under each number shows calcium dissolved per gram of cement; the second shows the percentage dissolved of the total calcium present (in 1 gram of cement). With 432 and 864 cubic centimeters of water, respectively, the solution continued to increase for a number of days, but with the other three volumes, the results were constant after the first twenty-four hours.

Table VII shows that from 87 to 92 per cent of the calcium in a cement will go into solution in twenty-four hours, provided the volume of water present is sufficiently large. Further it seems probable that all of the calcium would dissolve if a still greater volume were used, providing the cement were sufficiently fine and the last trace of carbon dioxide had been removed both from the cement and from the water. We can now see why such results as those of Hart [12] are misleading. Both the potassium and the sulphate ion may be found in solution, but, in place of being the chief constituent, they are negligible in quantity as compared with the calcium.

On plotting the percentage calcium dissolved against the volume of water present for each cement in Table VII, it may be

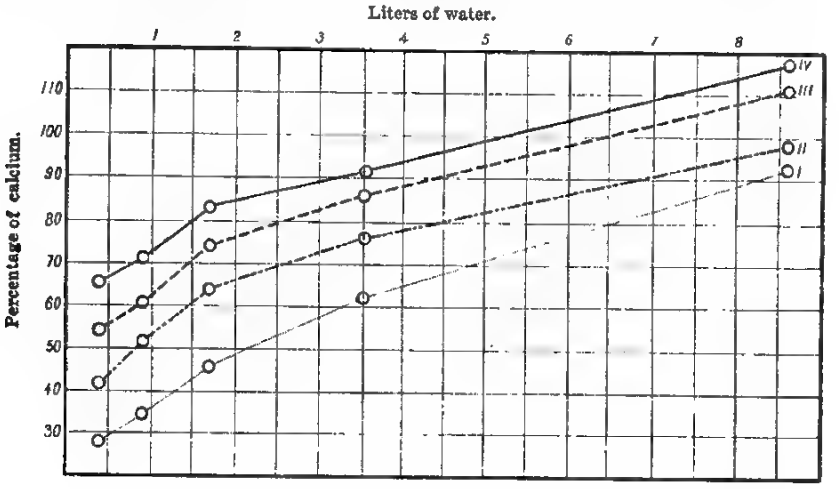

FIG. 1. Percentage of calcium dissolved in relation to volume of water present. In order to facilitate comparison, curve II is drawn 10 points above its true position; III, 20 points; and IV, 30 points.

seen that the curves agree very closely (fig. 1). This is somewhat surprising in that all the cements are of different manufacture. It will be noted on comparing Tables III and VII that the two cements having the highest percentage of calcium in solution are lowest in calcium. There seems to be no relation between the percentage solubility and the amounts of other constituents present.

INTERPRETATION OF THE RESULTS IN TERMS OF VARIOUS THEORIES OF HYDRATION

Unquestionably the most important recent advances in our knowledge of the constitution of cement have been accomplished

[12] Loc. cit.

by methods of microscopy and physical chemistry. However, we believe that the usefulness of methods of analytical chemistry has not been exhausted in this field and that such methods still offer points for attacking these problems, either alone or in conjunction with other methods. Although this work was undertaken simply with the object of learning what constituents of cement would dissolve in water under favorable conditions—and in what quantities—the results obtained are closely related to hydration phenomena. The amount of calcium hydroxide found in solution in presence of varying volumes of water can be hardly explained by the laws of solubility. The solution never reaches the point of saturation. This cannot be due to the other substances in solution, because the amounts are relatively too small. The nature of the solvent, the method of agitation, the kind of solute, and the temperature (within certain limits) all have been kept constant. Therefore the determining factor is evidently the formation of calcium hydroxide, by the hydration of the various compounds present in cement. Or it is the inhibition of hydration caused by calcium hydroxide in solution. This effect may be said to be twofold. It diminishes the speed of hydration and also the total amount of hydration possible under given conditions. Or, the speed of hydration is diminished until the amount taking place in twenty-four hours is too small to be detected by the methods of analysis employed.

The conditions under which cement is hydrated in this work are, of course, abnormal, as compared with conditions in practice. This comes about through the use of a large volume of water and through agitation, which keeps the granules separated and keeps a large surface exposed to the water. The exclusion of carbon dioxide probably does not constitute such a great variation from normal conditions as at first appears. Over the surface of newly placed concrete or mortar, a thin film of calcium carbonate forms almost immediately, and this protects the interior from further contact with this gas. Keeping these facts in mind, let us now try to interpret the results in terms of results that have been obtained by various investigators of the question of hydration of cement. In general, the agreement is striking, though there are some important differences. It may be also stated that not sufficient work has been done along these lines to justify the formulation of any new theory of hydration.

The theory of Richardson [18] is that—

> On addition of water to the stable system made up of the solid solu-

[18] Cf. Meade, Richard K., Portland Cement. The Chemical Publishing Co., Easton, Pa. (1911), 22.

tions which compose Portland cement, a new component is introduced, which immediately results in lack of equilibrium, which is only brought about again by the liberation of free lime. This free lime the moment that it is liberated is in solution in the water, but owing to the rapidity with which it is liberated from the aluminate, the water soon becomes supersaturated with calcic hydrate and the latter crystalizes out in a network of crystals, which binds the particles of undecomposed Portland cement together.

The results of the present work show that one of the products of hydration is undoubtedly calcium hydroxide and that the water present contains some of it in solution. Further it is well established that crystals of this compound are found in hardened cement. There is a question, however, whether or not these crystals are as important as the writer intimates and also as to the mechanism of their formation. Considering the amount of water that is ordinarily mixed with cement and the low solubility of calcium hydroxide, it is evident that if at a given instant all the water were saturated with this compound, and then all the calcium hydroxide should crystallize out, the percentage of the total calcium in the cement so affected would be small. For example, let us consider that one kilogram of cement is mixed with sufficient water to produce a paste of normal consistency. The average amount of water required is from 20 to 25 per cent of the weight, or say 250 cubic centimeters. Now cement will contain on an average of 62 per cent calcium oxide, or 620 grams—which is equivalent to approximately 819 grams of calcium hydroxide—per kilogram. Disregarding the portion of the water that enters into combination with calcium oxide and is unavailable for other reasons, the 250 cubic centimeters present would dissolve only 0.41 gram of the solid at a temperature of 20° C. according to Seidel.[14] This is, of course, on the assumption made previously that the relative amounts of other substances in solution are not sufficient materially to affect the solubility of calcium hydroxide. It can be seen that this amount of the substance is not sufficient to bind the cement together.

It is possible to consider that the crystallization occurs progressively, that is, when the solution becomes saturated, some of the dissolved hydroxide crystallizes out, more goes into solution as a result of further hydration, and so on. However, a number of facts are opposed to such a view. If the water is not saturated at a given time, and crystals of calcium hydroxide are present, it is more likely that some of these would dissolve than

[14] Loc. cit.

that more of the calcium compounds in the cement would be hydrated. This is on the basis of the results presented in this paper, which show that the presence of calcium hydroxide in solution tends to inhibit further hydration. Further, it is known that if an imperfect crystal is suspended in a saturated solution of the same substance, it does not change in weight, though it may change in form sufficiently to become again regular.

The principal components of cement are compounds of calcium with aluminium and of calcium with silicon. Indeed, according to Rankin:[15]

> Microscopical examination of commercial Portland cement clinker shows it to be made up largely (over 90 per cent) of the three compounds, $2CaO.SiO_2$, $3CaO.SiO_2$ and $3CaO.Al_2O_3$. It would therefore appear that the value of Portland cement as a cementing material when mixed with water is largely due to one or more of these compounds.

Now since about 90 per cent of the total calcium in a cement is found in solution after treating with water under given conditions, with indications that still more could be dissolved, it follows that (1) all the important compounds may be rapidly hydrated under favorable conditions and that (2) one product of the hydration is always calcium hydroxide.

The colloid theory for the setting of cement was advanced by Michaelis.[16] His idea is that the most important step is the formation of a gelatinous mass containing calcium oxide, silica, and water. Later this colloid dries and hardens, and to it is due the principal strength of the cement. Considerable work has been done by others on the basis of this theory, using cement itself or one of the calcium aluminates.

Schott[17] and Keiserman[18] found that, when certain calcium aluminates are hydrated, aluminium hydroxide is split off. Stern[19] found that aluminates were decomposed by water forming the hydroxide of calcium and aluminium. Later he dialyzed the filtrate and found that calcium passed the membrane, but with only a trace of aluminium. Klein and Phillips[20] repeated the work of Stern, taking great care to exclude carbon dioxide during the operation. They used tricalcium aluminate and found

[15] Rankin, George A., *Journ. Franklin Inst.* (1916), 181, 770.
[16] Michaelis, W., *Cement & Eng. News* (1909), 21, 298, 338.
[17] Schott, O., ibid. (1910), 22, 515.
[18] Keiserman, ibid. (1911), 23, 10.
[19] Stern, E., loc. cit.
[20] Klein, A. A., and Phillips, A. J., *Tech. Paper, U. S. Bur. Standards*, (1914), No. 43, 18.

that the liquid passing the membrane contained aluminium and calcium in about the original proportions. They conclude from this that no colloid is formed and that the substance is not broken up by hydration.

The work in this laboratory favors Stern's results, though it must be remembered that commercial cement was used in every case and not an aluminate alone. It may be also said that if a colloid forms according to Michaelis's theory it is broken up by a large excess of water, as the presence of such a large amount of dissolved calcium with only a trace of silicon (in any form) shows. Or the explanation may be that the colloid does not form because the concentration of the calcium hydroxide solution is not sufficiently high.[21]

It is generally conceded by cement investigators that the strength of a test specimen depends to some extent on the fineness of grinding; in fact there is no doubt that, other factors being equal, the finer a cement is ground the greater strength it will give mortar briquettes. A proof of this is that if specimens of hardened mortar or paste are reground the powder may be again mixed with water, and a fair degree of strength obtained.[22] The mass may be again ground, and water added, with a like result. The usual explanation offered for this is that during the first gauging the water cannot penetrate the larger particles of cement and that the cores of these remain unchanged. When reground and regauged, these parts become active, and there is sufficient new paste to cement the whole together and so on. The present work supports this explanation, but indicates that there are other factors to be considered. By referring to Tables IV and VII it may be seen that, although only about 40 per cent of the total calcium in the cements was hydrated and dissolved when agitated for fifteen days in the original experiment with fine cement, approximately 90 per cent of the calcium went into solution in only twenty-four hours, when the relative volume of water was increased twentyfold. Since the cement was of the same fineness in both cases, it may be seen that the volume of water is of importance as well as the size of the particles. Further it is probable that if the finest cement flour obtainable were gauged with water it would not be completely hydrated—not because of size of grain, but because of reasons already ex-

[21] Michaelis, loc. cit.
[22] Michaelis, loc. cit.

plained—and that if this material after hardening were reground another set could be obtained.

This leads us to the conclusion that the presence of more water when cement is gauged facilitates hydration and should, therefore, result in greater strength. This last is contrary to the general opinion on the subject. As a rule, especially for short periods, the addition of more water means lower strength [23] for briquettes. In concrete practice, very wet mixes are not recommended.[24]

Here again are other factors to be considered. The water that remains mixed with the concrete or mortar until setting is complete reduces the strength, because it decreases the density of the material and consequently the cohesion. The water that separates, either by leaking through the forms or rising to the top, carries calcium hydroxide, one of the products of hydration, in solution. Previous work by one of us [25] has indicated that an agency that removes dissolved calcium hydroxide or interferes with the cohesion will lower the strength. Therefore we believe that, although the strength is increased by the use of a higher percentage of water, other factors have a still greater tendency to lower the strength, and consequently the latter is the net result. A series of experiments just started indicates that this conclusion is correct, although sufficient data have not been obtained to justify any definite statement as yet. A series of mortar briquettes was made with gradually increasing amounts of water, starting with the amount calculated from normal consistency tests. There was a decrease in strength with increase of water. A second series was made with the same amounts of water, but, before a given mix was molded, it was placed in a metal vessel, and the water was evaporated until the weight showed that the amount indicated by the normal consistency tests was reached. It was assumed that the extra water temporarily present would facilitate hydration and dissolve more calcium hydroxide and that this hydroxide would remain in solution even after a portion of the water was evaporated, because the solution was not near the saturation point, even though the solubility of this compound decreases with a rise in temperature. After the evaporation each mix was immediately regauged and placed in molds. In general, the strength increased as the water increased, contrary to the first series.

[23] Cf. Larned, E. S., *Proc. Am. Soc. Test. Mat.* (1903), 3, 401.

[24] Cf. Taylor and Thompson, A Treatise on Concrete. John Wiley and Sons, New York (1917), 251.

[25] Witt, J. C., *This Journal, Sec. A* (1916), 11, 288.

SUMMARY

When cement is shaken with water in a closed vessel large amounts of calcium with relatively small amounts of most of the other elements present go into solution.

The factors that effect the results have been found to be (a) absence of carbon dioxide, (b) method of agitation, (c) fineness of grain, (d) volume of water, and (e) time. Of these, volume of water is the most important. The effect of temperature has not been studied.

As the volume of water is increased, the amount of calcium going into solution in a given time increases rapidly. When cement is treated with approximately eight thousand times its weight of water, 90 per cent of the calcium present goes into solution in twenty-four hours, with indications that still more would dissolve in a greater volume.

Though the work was not undertaken as a study of hydration, the results obtained are closely related to the theories of hydration that have been formulated from time to time.

Since all the important compounds in cement contain calcium, and 90 per cent of all calcium present goes into solution, it may be stated that under favorable conditions the hydration of all important compounds results in the formation of calcium hydroxide.

It has not been found possible to obtain a saturated solution of calcium hydroxide by shaking cement in water. This may be due to the fact that presence of dissolved calcium hydroxide inhibits further hydration, or it may be that when the concentration of the calcium hydroxide solution reaches a certain value a colloid is formed, according to Michaelis' theory.

ILLUSTRATION

TEXT FIGURE

FIG. 1. Percentage of calcium dissolved in relation to volume of water present.

PHILIPPINE ECONOMIC-PLANT DISEASES

By Otto A. Reinking

(*From the College of Agriculture, Los Baños*)

TWENTY-TWO PLATES AND FORTY-THREE TEXT FIGURES

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Physalospora guignardioides Sacc.
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Bacillus solanacearum Erw. Smith.
Erysiphaceae.
Phomopsis capsici (Magnaghi) Sacc.
Vermicularia capsici Syd.
Capsicum frutescens Linn. Red pepper.
Vermicularia capsici Syd.
Carica papaya Linn. Papaya.
Aspergillus periconioides Sacc.
Colletotrichum papayae (Henn.) Syd.
Didymella caricae Tassi.
Diplodia caricae Sacc.
Erysiphaceae.
Fusarium.
Fusarium heveae Henn.
Lasiodiplodia theobromae (Pat.) Griff. et Maubl.
Mycosphaerella caricae Syd.
Penicillium.
Phytophthora faberi Maubl.
Pythium debaryanum Hesse.
Rhizoctonia.
Rhizopus.
Citrus spp. Oranges, lemons, limes, pomelos.
Bark rot.
Chlorosis, nonparasitic.
Die-back, lack of nutrition.
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Rhizoctonia.
Citrus maxima (Burm.) Merr. (*C. decumana* Linn.).
Aschersonia sclerotoides Henn. (On coccids.)
Colletotrichum glocosporioides Penz.
Corticium salmonicolor Berk. et Broome.
Eutypella citricola Speg.
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Loranthus philippensis Cham. (Epiphytes.)
Meliola.
Micropeltis.
Mottled leaf, nonparasitic.
Nectria episphaeria (Tode.) Fr.
Penicillium.
Phyllosticta circumsepta Sacc.
Scaly bark.
Spiny mold, imperfect fungus.
Citrus nobilis Lour.
Cytospora aberrans Sacc.
Diaporthe citrincola Rehm.
Diplodia aurantii Catt.
Eutypella citricola Speg.
Hypoxylon atropurpureum Fr. (On coccids.)
Massarina raimundoi Rehm.
Tryblidiella mindanaensis Henn.
Tryblidiella rufula (Spreng.) Sacc.
Valsaria citri Rehm.
Zignoella nobilis Rehm.
Cocos nucifera Linn. Coconut.
Anthostomella cocoina Syd.
Bacillus coli (Escherich).
Bud rot, bacterial.
Capnodium footii Berk. et Desm.
Chaetosphaeria eximia Sacc.
Coniosporium dendriticum Sacc.
Coprinus fimbriatus B. et Br.
Coprinus friesii var. *obscurus* Pat.
Cytospora palmicola B. et C.
Diplodia cococarpa Sacc.
Diplodia cococarpa var. *malaccensis* Tassi.
Diplodia epicocos Cooke.
Diplodia epicocos Cooke var. *minuscula* Sacc.
Elfvingia tornata (Pers.) Murr.
Eutypella cocos Ferd. et Winge.
Exosporium durum Sacc.
Ganoderma incrassatum (Berk.) Bres. var. *substipitata* Bres.
Gloeoglossum glutinosum (Per.) Durant.
Hormodendron cladosporioides (Fr.) Sacc.
Palawania cocos Syd.
Peroneutypella cocoes Syd.

Cocos nucifera Linn.—Continued.
Pestalozzia palmarum Cke. et Grev.
Phyllosticta cocophylla Pass.
Rosellinia cocoes Henn.
Sterility of nuts.
Coffea spp. Coffee.
Aithaloderma longisetum Syd.
Coniothyrium coffeae Henn.
Foot rot.
Hemileia vastatrix B. et Br.
Micropeltis mucosa Syd.
Rhizoctonia.
Sclerotium.
Colocasia esculentum Schott (*C. antiquorum* Schott). Gabi.
Phytophthora colocasiae Rac.
Cucumis sativas Linn. Cucumbers.
Cercospora.
Plasmopara cubensis (B. et C.) Humphrey.
Cucurbita maxima Duch. Calabaza, squash.
Erysiphaceae.
Plasmopara cubensis (B. et C.) Humphrey.
Daucus carota Linn. Carrot.
Rhizoctonia.
Dioscorea esculenta (Lour.) Burkill. Yams.
Cercospora pachyderma Syd.
Cercospora ubi Rac.
Ellisiodothis rehmiana Theiss et Syd.
Gloeosporium macrophomoides Sacc.
Lasiodiplodia theobromae (Pat.) Griff. et Maubl.
Mycosphaerella dioscoreicola Syd.
Phoma oleracea Sacc.
Phomopsis dioscoreae Sacc.
Phyllachora dioscoreae Schwein.
Phyllachora rehmiana Theiss. et Syd.
Phyllosticta graffiana Sacc.
Rhizopus.
Uredo dioscoreae (Berk. et Brm.) Petch.
Uredo dioscoreae-alatae Racib.
Dolichos lablab Linn. Lablab bean.
Cercospora.
Didymella lussoniensis Sacc.
Diplodia lablab Sacc.
Dolichos lablab Linn.—Continued.
Septoria lablabina Sacc.
Septoria lablabis Henn.
Vermicularia horridula Sacc.
Woroninella dolichi (Cke.) Syd.
Ficus carica Linn. Fig.
Kuehneola fici (Cast.) Butl.
Phyllachora. On wild figs.
Uredo fici Cast.
Glycine max (Linn.) Merr. (*G. hispida* Maxim.). Soy bean, soja.
Peronospora.
Rhizoctonia.
Trotteria venturioides Sacc.
Uromyces sojae Syd.
Gossypium spp. Cotton.
Bacterium malvacearum Erw. Smith.
Kuehneola desmium (B. et Br.) Syd.
Uredo desmium (B. et Br.) Petch.
Hevea brasiliensis (HBK) Muell.-Arg. Para rubber.
Eutypella heveae Yates.
Fomes lignosus (Kl.) Bres.
Helminthosporium heveae Petch.
Megalonectria pseudotrichia (Schw.) Speg.
Physiological Trouble.
Phytophthora faberi Maubl.
Spotting of prepared plantation rubber, saprophytic fungi.
Tryblidiella mindanaensis Henn.
Hibiscus sabdariffa Linn. Roselle.
Phoma sabdariffae Sacc.
Ipomoea batatas Poir. Sweet potato.
Lasiodiplodia theobromae (Pat.) Griff. et Maubl.
Rhizopus.
Lactuca sativa Linn. Lettuce.
Tipburn, nonparasitic.
Lycopersicum esculentum Mill. Tomato.
Bacillus solanacearum Erw. Smith.
Erysiphaceae.
Pythium debaryanum Hesse.
Rhizoctonia.
Mangifera indica Linn. Mango.
Cercospora mangiferae Koord.
Endoxyla mangiferae Henn.

Mangifera indica Linn.—Continued.
Leptothyrium circumscissum Syd.
Meliola mangiferae Earle.
Pestalozzia funerea Desm.
Pestalozzia pauciseta Sacc.
Phyllachora sp.
Manihot dichotoma Ule. Ceara rubber.
Phyllosticta manihoticola Syd.
Manihot utilissima Pohl. Cassava, camoting cahoy.
Cercospora henningsii Allesch.
Cercospora manihotis Henn.
Colletotrichum lussoniense Sacc.
Diplodia manihoti Sacc.
Guignardia manihoti Sacc.
Guignardia manihoti Sacc. var. *diminuta* Sacc.
Phoma herbarum Westd.
Steirochaete lussoniensis Sacc.
Morus alba Linn. Mulberry.
Botryodiplodia anceps Sacc. et Syd.
Diplodia mori West.
Kuehneola fici (Cast.) Butl. var. *moricola* Henn.
Phyllactinia suffulta (Reb.) Sacc.
Traversoa dothiorelloides Sacc. et Syd.
Twig fungi.
Valsaria insitiva (de Not.) Ces. et de Not.
Mucuna deeringiana Merr. (*Stizolobium deeringiana* Bort). Velvet bean.
Cercospora stizolobii Syd.
Uromyces mucunae Rabh.
Musa sapientum Linn. Banana.
Bacterial stem rot.
Diplodia crebra Sacc.
Fruit blast.
Macrophoma musae (Cke.) Berl. et Vogl.
Mycosphaerella musae Speg.
Plicaria bananincola Rehm.
Sporodesmium bakeri Syd.
Musa textilis Née. Abacá.
Bacterial heart rot.
Macrophoma musae (Cke.) Berl. et Vogl.
Mycosphaerella musae Speg.
Nicotiana tabacum Linn. Tobacco.
Nicotiana tabacum Linn.—Cont.
Bacillus solanacearum Erw. Smith.
Bacterial blight.
Cercospora nicotianae Ell. et Ev.
Chlorosis.
Curing and fermenting troubles.
Leaf spotting.
Fusarium.
Heterodera radicicola Greef et Müller. (Nematodes.)
Phytophthora nicotianae Breda de Haan.
Pythium debaryanum Hesse.
Rhizoctonia.
Sclerotium.
Oryza sativa Linn. Rice.
Bacterial leaf stripe.
Calonectria perpusilla Sacc.
Cercospora.
Clasterosporium punctiforme Sacc.
Coniosporium oryzinum Sacc.
Entyloma oryzae Syd.
Haplographium chlorocephalum (Fres.) Grove.
Helminthosporium.
Leptosphaeria (*Leptosphaerella*) *oryzina* Sacc.
Myrothecium oryzae Sacc.
Oospora oryzetorum Sacc.
Ophiobolus oryzinus Sacc.
Phyllosticta glumarum Sacc.
Phyllosticta miurai Miyake.
Rhizoctonia.
Sclerotium.
Septoria miyakei Sacc.
Sordaria oryzeti Sacc.
Spegazzinia ornata Sacc.
Straight or sterile head.
Ustilaginoidea virens (Cke.) Tak.
Pachyrrhizus erosus (Linn.) Urb. (*P. angulatus* Rich.). Sincamas.
Phakospora pachyrhizi Syd.
Phaseolus spp. Beans.
Cercospora lussoniensis Sacc.
Erysiphaceae.
Phyllachora phaseolina Syd.
Pseudomonas phaseoli Erw. Smith.
Rhizoctonia.
Sclerotium.

Phaseolus spp.—Continued.
Sooty mold.
Uromyces appendiculatus (Pers.) Lk.
Phaseolus lunatus Linn.
Cladosporium herbarum (Pers.) Lk.
Diplodia phaseolina Sacc.
Phaseolus vulgaris Linn.
Asteroma phaseoli Brun.
Diplodia phaseolina Sacc.
Piper betle Linn. Icmo, betel pepper.
Oospora perpusilla Sacc.
Pisum sativum Linn. Pea.
Erysiphaceae.
Psophocarpus tetragonolobus DC. Winged bean, calamismis.
Woroninella psophocarpi Rac.
Raphanus sativus Linn. Radish.
Bacillus carotovorus Jones.
Saccharum officinarum Linn. Sugar cane.
Aeginetia indica Linn. (Broom rape.)
Apiospora camptospora Penz. et Sacc.
Bakerophoma sacchari Diedicke.
Cercospora.
Coniosporium extremorum Syd.
Coniosporium vinosum (B. et C.) Sacc.
Dictyophora phalloidea Desvaux.
Haplosporella melanconioides Sacc. forma.
Heterodera radicicola Greef et Müller. (Nematodes.)
Marasmius.
Melanconium lineolatum Sacc.
Melanconium sacchari Massee.
Meliola arundinis Pat.
Phyllachora sacchari Henn.
Puccinia kuehnii (Krueg.) Butl. [*Uredo kuehnii* (Krueg.) Wakk. et Went].
Rhizoctonia.
Sereh disease.
Stem rot, bacterial.
Ustilago sacchari Rabh.
Saccharum spontaneum Linn. Wild sugar cane.
Haplosporella melanconioides Sacc.
Saccharum spontaneum Linn.—Cont.
Phyllachora sacchari spontanei Syd.
Ustilago sacchari Rabh.
Sesamum indicum Linn. Sesame, linga.
Cercospora sesami A. Zimm.
Erysiphaceae.
Gloeosporium macrophomoides Sacc.
Helminthosporium sesameum Sacc.
Phoma sesamina Sacc.
Vermicularia sesamina Sacc.
Solanum melongena Linn. Eggplant.
Bacillus solanacearum Erw. Smith.
Diplodina degenerans Diedicke.
Gloeosporium melongenae Sacc.
Phoma solanophila Oud.
Sarcinella raimundoi Sacc.
Solanum tuberosum Linn. Potato.
Bacillus phytophthorus Appel.
Bacillus solanacearum Erw. Smith.
Phytophthora infestans (Mont.) de Bary.
Theobroma cacao Linn. Cacao.
Aspergillus delacroixii Sacc. et Syd.
Botryosphaeria minuscula Sacc.
Canker.
Cyphella holstii Henn.
Die-back.
Fusarium theobromae App. et Strunk.
Lasiodiplodia theobromae (Pat.) Griff. et Maubl.
Lichens.
Mycogone cervina Ditm. var. *theobromae* Sacc.
Nectria bainii Massee var. *hypoleuca* Sacc.
Nectria discophora Mont.
Oospora candidula Sacc.
Ophionectria theobromae (Pat.) Duss.
Physalospora affinis Sacc.
Phytophthora faberi Maubl.
Vigna spp. Cowpeas.
Cercospora.
Erysiphaceae.

INTRODUCTION

Fungous diseases are found on practically all cultivated and wild plants in Laguna Province, Philippine Islands. From this local abundance it is to be presumed with a great degree of assurance that they are equally prevalent in most, if not all, the other agricultural regions of the Islands. They are often the limiting factors in the raising of many agricultural crops. Climatic conditions of the Philippines account for the great number and destructiveness of plant diseases, for the growth and development of fungi are enhanced by warmth and moisture. During the rainy season both of these factors are present, thereby aiding the large destruction during this period of the year. Plant diseases are seasonal; that is, they are more numerous and severe during the wetter months of the year, extending from July to November. A person going through the Islands during the dry season will not be impressed with the number and destructiveness of plant diseases, but during the rainy season the reverse will be found true. No complete estimates of losses due to plant diseases have been prepared in the Philippines, but it would be safe to say that in this section of the country at least 10 per cent of agricultural crops are destroyed by fungi.

Certain articles on phytopathology in the tropics give an en-

tirely wrong impression of the number and destructiveness of the diseases.[1] In the Malayan regions, at least so far as the Philippines are concerned, there are represented all the groups of fungi that are present in temperate regions. Extremely destructive diseases are produced by some members of each group. Forest pathology has never been really investigated, but there are many important and destructive timber fungi. The powdery mildews, Erysiphaceae, may be very abundant and often destructive during the cooler, drier months of the year. The perfect stage has been only observed with a powdery mildew growing on the leaves of a forest tree, *Premna cumingiana* Schau. This ascigerous stage is of the genus *Uncinula*. Leaf-spotting fungi are very common and some are extremely destructive. Destructive rusts are present on coffee, sugar cane, and sorghum. Bacterial diseases are present in abundance, many being highly destructive. Certain diseases caused by Phycomycetes and imperfect fungi may be very severe. There are as many destructive plant diseases in the Philippine Islands as there are in the United States, if there are not more.

The seriousness of some of the diseases can be judged by the fact that the coffee industry of the Islands was wiped out by a fungus, that the coconut industry suffers severely in certain sections from destruction of trees in all stages of growth due to bud rot, that the abacá industry sustains great losses due to bacterial attack, that one-half of the cacao fruit is destroyed by fungi, and that rice culture is seriously hampered by fungus attacks. This is also true of the sugar and citrus industries and the culture of all vegetables.

The great factors in the spread and destructiveness of fungi are the lack of proper culture, of sanitation, of pruning, and of spraying. The Filipino farmer plants his crops and allows Providence to do the rest. Ignorance concerning plant diseases and disease control, together with lack of foresight of the people, along general cultural lines, accounts for a good deal of loss. In some few instances growers know that the plants are diseased and that they ought to be removed, but still they do nothing. They figure that as long as they are getting fair returns from their crops they need not worry about the future. There is great need of education among the mass of Filipino farmers with regard to the spread of plant diseases and their prevention as well as for providing properly educated inspectors to safe-

[1] Westerdijk, Phytopathology in the tropics, *Ann. Missouri Bot. Gardens* (1915), 2, 307-313.

guard the interests of the thrifty and foresighted farmer who does know how to spray and who puts his knowledge into practice. As it is, practically no spraying is carried on in the Islands.

This paper has been written in order to give some idea of the prevalence of plant diseases, their causes, mode of attack, plant hosts, amount of damage, and methods of control. While the list of diseases is by no means complete and while it takes into consideration primarily those diseases found in Laguna and near-by provinces in Luzon, it will demonstrate that practically all agricultural crops have their fungous enemies. Many of these diseases are due to fungus species new to science. The contribution of these new species has been largely due to the collections of Prof. C. F. Baker, professor of agronomy in the College of Agriculture.

ANANAS COMOSUS (LINN.) MERR. (A. SATIVUS SCHULTES F.). PINEAPPLE

LEAF SPOT: ASTERINELLA STUHLMANNI (HENN.) THEISSEN

Symptoms.—The lower leaves of the pineapple are frequently and sometimes seriously attacked by this superficial leaf-spotting fungus. The black mass of mycelium produces spots that extend rapidly and often cover the entire leaf. Older spots are frequently elevated, due to the shrinkage of the surrounding tissue, and they have dark gray centers covered with minute black specks, the perithecia. The fungus causes the premature death of the lower older leaves.

Causal organism.—The perithecia are usually seen with the naked eye. They appear as minute black specks in the grayish diseased portion. The asci within the perithecia are sack-shaped bodies and usually contain eight ascospores. The latter are two-celled and elongated, with a large vacuole in each cell. The fungus is a superficial grower, but feeds on the cells by the production of haustoria, and in this way it weakens the leaves.

Control.—Sanitation methods are advisable, such as the collection and destruction of the older, badly diseased leaves. In severe cases of infection crop rotation should be practiced.

Lembosia bromeliacearum Rehm. is also found growing superficially on the living leaves, parasitizing them by the production of haustoria.

SOOTY MOLD

Symptoms.—Black felty masses of a superficially growing fungus may be produced on the under surfaces of leaves. The

fungus has not been prevalent enough to cause any serious damage (Plate XIV, fig. 1). It has not been identified.

Steirochaete ananassae Sacc. and *Diplodia ananassae* Sacc. are found on dead leaves.

ANDROPOGON SORGHUM LINN. (SORGHUM VULGARE PERS.). SORGHUMS, KAFFIRS, MILOS

GRAIN MOLD: HELMINTHOSPORIUM CARYOPSIDUM SACCARDO

Symptoms.—Grains are frequently covered with a dense black or sometimes dark greenish mold. Generally little damage is done, but in severe cases of infection seeds may be destroyed.

Causal organism.—The mold is made up of mycelium, conidiophores, and the many-celled, curved brownish conidia.

Control.—The seeds should be carefully dried and stored in a well-ventilated dry place.

KERNEL SMUT: USTILAGO SORGHI (LK.) PASSARINI

Symptoms.—This disease though not serious is, however, occasionally present. Individual grains of the panicle are affected. Diseased heads appear normal except for the infected grains. Smutted grains are much enlarged and have a black smutty mass of spores protruding between the glumes (Plate I, fig. 3).

Causal organism.—The smutty mass is composed of round, smooth, brownish smut spores. These spores germinate by the production of a promycelium, from which are produced hyaline sporidia.

Control.—Only seeds free from smut should be planted. All diseased heads should be collected and burned. Crop rotation will check the disease.

LEAF SPOT: PHYLLACHORA SORGHI V. HÖHNEL

Symptoms.—Leaves are badly attacked by this fungus, which produces thickly scattered black spots over the surface. Spots are small, 1 to 4 millimeters in diameter, roundish, sometimes elongated, raised, extending through the leaf on both surfaces, and are made up of hard stromatic masses of the fungus (Plate I, fig. 1). These black stromatic masses may be surrounded by a dark reddish or yellowish ring, produced by the discoloration of leaf tissue. The reddish spots frequently run together, producing a much-reddened leaf. The disease is often serious enough to destroy leaves for use as fodder, as well as to lower the vitality of plants.

Causal organism.—Within the stromata are produced usually one or two perithecia, which contain numerous asci, ascospores,

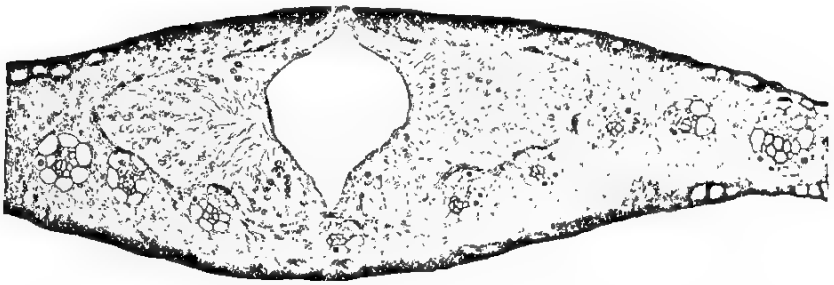

FIG. 1. *Phyllachora sorghi* v. Höhnel. Cross section of stroma, showing perithecium, ostiolum, asci, and ascospores (× 75). Vascular bundles of leaf develop normally within the mass of fungus mycelium.

and paraphyses. Sections through the stromata disclose the interesting fact that the vascular bundles of the leaf are not at all injured, for these bundles develop apparently normally within the mass of fungus mycelium (fig. 1). The passage of food and water is not inhibited by the fungus, but the vitality of the plant is lowered, for the fungus absorbs food for the development of its own body and also reduces the chlorophyll area of the leaf. Asci are typical, club-shaped bodies containing usually eight hyaline spores. The ascospores are elongated and granular, with the contents often collected in each end, which in some cases makes them appear two-celled (fig. 2). The paraphyses are slender, hyaline bodies and are produced in abundance.

Control.—No special control need be practiced. Crop rotation and sanitation will check the disease.

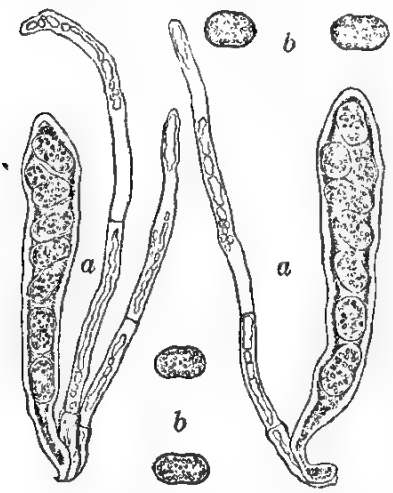

FIG. 2. *Phyllachora sorghi* v. Höhnel. *a*, asci with paraphyses (× 325); *b*, ascospores (× 325).

RUST: PUCCINIA PURPUREA COOKE

Symptoms.—Leaves may be entirely covered with rust sori, which lower the vitality of the plants and render them worthless for forage. Sori are brownish, at first closed, later ruptured, exposing the spores; are raised, elongated, about 1 millimeter by 2 millimeters, and are frequently surrounded by a dark reddish to purplish discoloration of the leaf surface. Badly infected leaves are usually entirely spotted and are nearly covered with a reddish to purplish discoloration (Plate I, fig. 2).

Causal organism.—Within the sori are produced in abundance one-celled, yellow to brown, usually ovate, spiny uredospores. They may in some cases retain a stalk. Prominent pores are developed. Teleutospores are not produced in such abundance. They may be developed along with uredospores, but usually predominate in sori within which they are found. The teleutospores are two-celled, thick-walled, dark brown, and smooth and usually have a stalk (fig. 3).

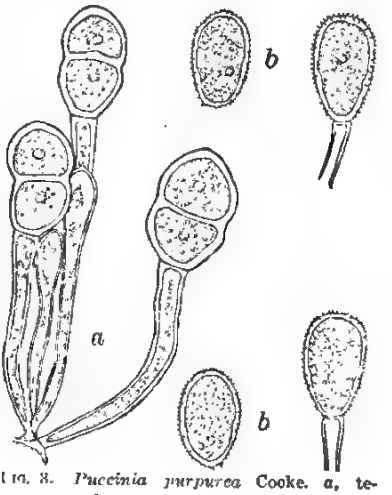

FIG. 3. *Puccinia purpurea* Cooke. *a*, teleutospores (× 315); *b*, uredospores (× 315).

Control.—Crop rotation and the destruction of badly diseased plants should be practiced.

SOOTY MOLD

Symptoms.—Frequently a dense sooty mold may be produced on leaves attacked by aphids. The fungus grows superficially, living on the exudate of the aphids. Little injury is done. The fungus has not been identified.

Didymosphaeria anisomera Sacc. has been identified from languished and dead leaves. On dying leaves, *Fumago vagans* Pers. may be found. *Coniosporium sorghi* Sacc. is found in dead and decaying stalks.

ANNONA MURICATA LINN. SOURSOP, GUANABANO

LEAF SPOT: PHYLLOSTICTA INSULARUM SACCARDO

Symptoms.—A common and sometimes destructive leaf disease. Spots are irregular and gray to whitish and start at the margins.

APIUM GRAVEOLENS LINN. CELERY

EARLY BLIGHT: CERCOSPORA APII FRIES

Symptoms.—Irregular roundish spots, which often run together forming blotches, may cover the leaf surface. When young the spots are light brownish, bordered with a yellowing of the leaf. Older spots have ashen gray centers surrounded with

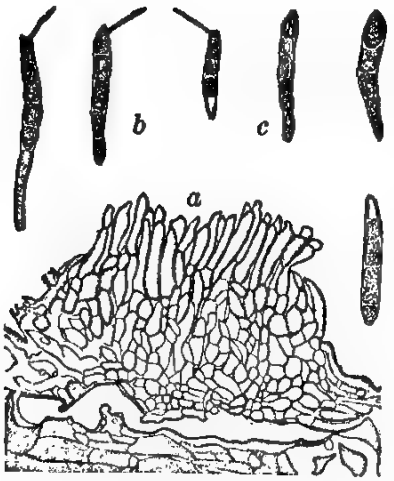

FIG. 4. *Septogloeum arachidis* Rac. *a*, cushionlike structure of conidiophores (× 350); *b*, germinating conidia (× 350); *c*, conidia (× 350).

brown. On the surface in the grayish portion is produced a black powdery mass.

Causal organism.—This black powdery mass is made up of hyaline, many-celled tapering conidia, which are produced on brownish conidiophores. The conidiophores are formed in groups and are septate.

Control.—Diseased plants should not be allowed to accumulate in the soil. Crop rotation should be practiced. In severe cases of infection, spraying Bordeaux mixture will have to be resorted to.

ARACHIS HYPOGAEA LINN. PEANUT, MANI

LEAF SPOT: SEPTOGLOEUM ARACHIDIS RACIBORSKI

Symptoms.—This well-known and widely distributed leaf spot may be extremely destructive on certain varieties of peanuts. It affects the lower leaves of the plant, and complete defoliation of this portion may result. From the lower portions the disease spreads to the upper leaves. The disease is most severe during damp weather, when both leaves and stems are attacked. Spots on the leaves are usually circular, black to brown, with a yellowish discoloration of the leaf tissue adjacent to the spot. The centers of older spots, chiefly on the under leaf surface, are specked with the raised masses of conidia and conidiophores. Spots on the stem are similar, but are usually elongated lengthwise. Certain varieties of peanuts show a marked degree of resistance.

Causal organism.—The more or less powdery, elevated bodies on the under surface of the spot are cushionlike structures made up of a mass of conidiophores and conidia (fig. 4). The elongated spores are brown and usually consist of from three to four cells. They germinate readily in water by the production of germ tubes, most frequently from one of the end cells (fig. 4). Inoculation experiments are easily carried out by spraying plants with a spore suspension. Penetration into the tissue is by means of the stomata (fig. 5). After gaining entrance, the mycelium spreads in local spots throughout the leaf, causing the death of the cells and the consequent browning of the tissue. The fungus

threads accumulate usually at the lower surface of the spots, producing the cushions of conidiophores and conidia. In pure culture it grows very slowly. On potato agar a raised, more or less leathery, dark brown mass of mycelium is produced. As yet no spores have been observed growing in pure culture.

Control.—The disease may be held in check by the growth of resistant and acclimatized varieties. The leaf spotting is most severe on the lower leaves, indicating infection from spores in the soil. Crop rotation will eliminate this last source of infection to a marked degree.

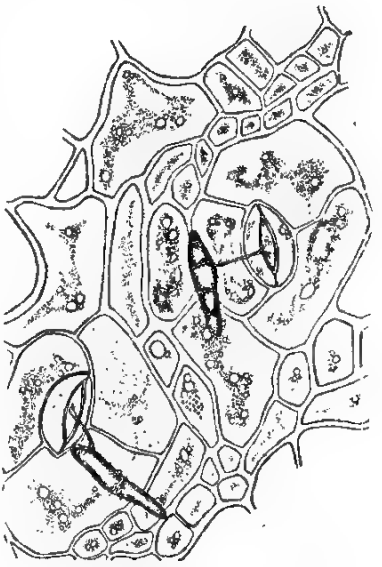

FIG. 5. *Septogloeum arachidis* Rac. Germinating conidia (× 350); germ tubes entering host tissue by way of stomata.

ROOT ROT: SCLEROTIUM

Symptoms.—Frequently peanuts are attacked by a fungus causing a rot of the root and the lower stems. Sclerotial bodies are always associated with the disease. As a rule the disease does not cause serious damage.

Causal organism.—The organism is a common soil fungus attacking a large number of plants. It is similar to that discussed under coffee.

Control.—Crop rotation should be practiced.

ARECA CATECHU LINN. BUNGA, BETEL PALM

The betel palm is attacked by a large number of fungi. *Pestalozzia palmarum* Cooke, *Exosporium pulchellum* Sacc., and *Exosporium hypoxyloides* Syd. cause leaf spots similar to those discussed under coconut. On dead leaves may be found *Guignardia arecae* Sacc., *Diplodia arecina* Sacc., and *Phomopsis palmicola* (Wint.) Sacc. On dead leaf sheafs may be found *Colletotrichum arecae* Syd., *Gloeosporium palmarum* Oud., and *Zygosporium oscheoides* Mont. On dead petioles may be found *Phomopsis arecae* Syd. and *Anthostomella arecae* Rehm. On dead fruit may be found *Gloeosporium catechu* Syd. On dead trunks may

be found *Peroneutypella arecae* Syd., *Eutypella rehmiana* (Henn. et Nym.) v. Höhnel, *Elfvingia tornata* (Pers.) Murr., and *Phellostroma hypoxyloides* Syd.

ARTOCARPUS COMMUNIS FORST. (ARTOCARPUS INCISA LINN. F.). BREADFRUIT

FRUIT ROT: RHIZOPUS ARTOCARPI RACIBORSKI

Symptoms.—The same fruit rot occurs on *Artocarpus communis* Forst. as is discussed under *Artocarpus integra* (Raderm.) Merr.

LEAF SPOT: CERCOSPORA ARTOCARPI SYDOW

Symptoms.—The common breadfruit tree is attacked by this typical *Cercospora* spot-producing fungus. Spots are more or less irregular with gray centers. Little damage is done.

Marchalia constellata (B. et Br.) Sacc. also causes a leaf spot. *Diplodia artocarpi* Sacc. may be found on languishing leaves. *Cycloderma depressum* Pat. may be found on the trunk.

ARTOCARPUS INTEGRA (RADERM.) MERR. (ARTOCARPUS INTEGRIFOLIA LINN. F.). JACK FRUIT, NANGCA

FRUIT ROT: RHIZOPUS ARTOCARPI RACIBORSKI

Symptoms.—The male inflorescence and young fruit may be attacked by this fungus. The blossoms are killed. Young inflorescences, 5 to 10 centimeters long, are subject to attack. On these the organism usually starts at the stem end or in wounds, causing a soft rot. The entire rotted portion is eventually covered with a dense black growth of the fungus, with the characteristic mold sporangia protruding. The fungus gradually advances, until the entire inflorescence becomes rotted and drops (Plate XIX, fig. 6). Extensive damage may be produced.

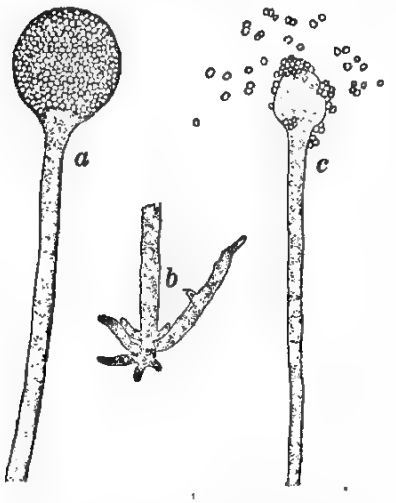

FIG. 6. *Rhizopus artocarpi* Rac. *a*, sporangium with spores (× 330); *b*, rhizoid (× 330), from tissue of fruit; *c*, bursted sporangium showing columella, sporangiophore, and spores (× 330).

Causal organism.—Typical *Rhizopus* sporangia and sporangiophores are produced. The outer walls of the sporangia are very delicate, breaking upon contact with water and spreading the spores (fig. 6). The fungus grows well in pure culture,

producing on potato agar a dense mass of sporangiophores with their blackish sporangia. Inoculation experiments prove this fungus to be highly parasitic. Young inflorescences on the tree are completely covered with the black mass of spore-bearing bodies three days after inoculation. The mycelium invades the tissue with rhizoids and produces a soft rot. The disease spreads rapidly during damp weather.

Control.—All diseased inflorescences should be carefully picked from the tree and the ground and destroyed. Care should be taken not to scatter the spores. In severe cases spraying with Bordeaux mixture may be practiced.

Dying leaves of the jack fruit may be attacked by *Diplodia artocarpina* Sacc. and *Dichotomella areolata* Sacc.

BETA VULGARIS LINN. CHARD

LEAF SPOT: CERCOSPORA

Symptoms.—The common leaf spot of the chard is often very destructive. Leaves of Swiss chard may be entirely covered with the characteristic spots. Spots when young are small and brownish to black; as they get older, they become larger, sometimes increasing up to 5 millimeters in diameter. Older spots are circular and brownish and may exhibit concentric rings, and the very oldest spots have an ashen-gray center bordered with a brownish ring. Spots may coalesce and cover nearly the entire leaf surface (Plate II, fig. 3).

Causal organism.—Conidiophores and conidia are produced in abundance in the ashen-gray center of the spots. Conidia are long, tapering, and hyaline; conidiophores are yellowish and in groups. The fungus grows readily in pure culture, producing on potato agar a more or less feltlike mass of white fungus, with a slight pinkish tinge.

Control.—The most satisfactory control consists in the collection and the destruction of diseased leaves and in crop rotation.

BRASSICA OLERACEA LINN. CABBAGE

BLACK ROT: PSEUDOMONAS CAMPESTRIS (PAMMEL.) ERW. SMITH

Symptoms.—The disease is characterized by the yellowing of the leaves at the margins and between the veins and the blackening of the veins. Cross sections of diseased petioles show blackened fibrovascular bundles (Plate X, fig. 2).

Causal organism.—Pure cultures of the bacteria indicate that the organism is the same as that attacking cabbage in the United States, whence it was undoubtedly introduced on seed.

The bacteria gain entrance into the plant through water pores at the margin of the leaf and through injuries on the leaf surface. After gaining entrance, the organism multiplies rapidly and spreads primarily through the fibrovascular bundles, causing them to blacken. The bacteria frequently ooze in a yellow mass from the cut bundles. From the leaves the organism spreads through the vascular bundles into the stem, causing a rot and consequent death of the plant (Plate X, fig. 2).

Control.—The collection and destruction of infected leaves may be effective as a control, if these leaves be picked before the organism has spread into the stem of the plant. When once the soil has become infected, crop rotation is the only method of control. Care should be taken that only healthy, noninfected seedlings are set out from the seed bed. The disease is spread on seeds. Seed treatment with either mercuric bichloride, 1 to 1,000, for fifteen minutes or 1 to 2 per cent formalin for twenty minutes is effective.

BRASSICA PEKINENSIS (LOUR.) SKEELS. PECHAY

LEAF SPOT: CERCOSPORA BRASSICICOLA P. HENNINGS

Symptoms.—Frequently severe spotting of the lower leaves occurs, making them unfit for food. Characteristic *Cercospora* spots, with ashen-gray centers bordered with light brown, are produced. These spots range from 1 to 15 millimeters in diameter. The older, larger spots frequently have concentric rings of gray and dark brown. The ashen-gray center of older spots is covered with a black mass of conidiophores and conidia (Plate II, figs. 1 and 2).

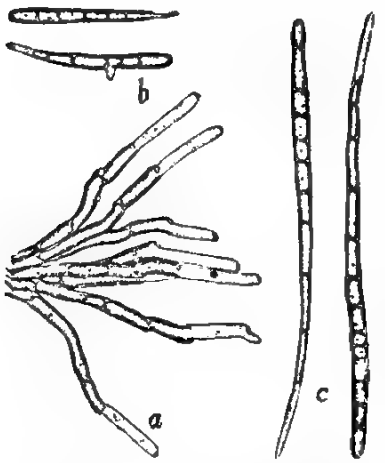

FIG. 7. *Cercospora brassicicola* Henn. *a*, group of conidiophores (× 340); *b*, small conidia germinating (× 340); *c*, typical needlelike conidia (× 340).

Causal organism.—The conidiophores are produced in groups arising from the stomata. They are septate and light brown. The conidia are hyaline, tapering, and from five- to fifteen-celled (fig. 7). Conidiophores as well as conidia may germinate and cause infection.

Control.—All diseased leaves should be collected and burned. Crop rotation should be practiced.

Cercospora armoraciae Sacc. also has been found on *Brassica*

pekinensis (Lour.) Skeels, where it produces a leaf spot similar to the one described above.

CANAVALIA GLADIATA DC., CANAVALIA ENSIFORMIS DC. HORSE BEANS, SWORD BEANS

These two beans may be attacked by *Elsinoe canavaliae* Rac., *Gloeosporium canavaliae* Syd., *Physalospora guignardioides* Sacc., and *Cercospora canavaliae* Syd. On decaying leaves of *Canavalia gladiata* DC. may be found *Didymium squamulosum* (Alb. et Schw.) Fr.

CAPSICUM ANNUUM LINN. RED PEPPER

BACTERIAL WILT: BACILLUS SOLANACEARUM ERW. SMITH

The bacterial wilt, which is so destructive on other solanaceous plants, attacks the peppers also. This disease is similar to that on tomato and tobacco, under which it is more fully described.

FRUIT ROT: VERMICULARIA CAPSICI SYDOW

Symptoms.—A spotting of the fruit characterized by the production of soft, often circular, sunken spots. The center of spots may dry, forming concentric rings within which small black spore-bearing bodies are produced. The disease is common, causing rotting of the fruit (Plate XVIII, fig. 2).

Causal organism.—The minute black specks produced in the depressed areas are the pycnidia of the fungus. They have numerous slender pointed setæ and produce elongated, hyaline conidia.

Control.—The collection and destruction of diseased pods should be practiced to check the disease. Spraying with Bordeaux mixture is effective when practicable.

On dried pods may be found the fungus *Phomopsis capsici* (Magnaghi) Sacc.

POWDERY MILDEW: ERYSIPHACEAE

Symptoms.—A white powdery growth may be produced on the surface of the leaves. At times the disease may be severe.

Causal organism.—The conidia are somewhat more elongated than the typical erysiphaceous spores, but they are produced in chains on the typical conidiophores.

Control—Badly diseased plants should be dusted with sulphur or sprayed with a standard fungicide. Crop rotation should be practiced.

CAPSICUM FRUTESCENS LINN. RED PEPPER

FRUIT ROT: VERMICULARIA CAPSICI SYDOW

Symptoms.—A fruit rot similar to that found on *Capsicum annuum* Linn.

CARICA PAPAYA LINN. PAPAYA

DAMPING-OFF: RHIZOCTONIA AND PYTHIUM DEBARYANUM HESSE

Symptoms.—Frequently young seedlings are attacked by soil fungi just at the surface of the ground. The stem first becomes watery, then turns brownish, and shrivels up, resulting in the falling over of the plant.

Causal organism.—Either of two common soil fungi, *Rhizoctonia* and *Pythium debaryanum* Hesse, may produce the disease. The *Rhizoctonia* grows well in pure culture, producing a brownish mycelium and brown sclerotial bodies. *Pythium* may be recognized in the plant tissue by its characteristic fruiting bodies.

Control.—All soil used for the growth of seedlings should be sterilized. Seed flats should be placed in a well-aërated place and sunned from time to time.

FRUIT ROT: FUSARIUM

Symptoms.—Frequently a *Fusarium* causes the rotting of mature fruit. The rot is similar in appearance to that caused by *Phytophthora*, except that the surface of this rot is covered with the dense growth of *Fusarium*. Spores are produced in abundance. Often rots are accompanied by various mold fungi, among them being a *Rhizopus* and a *Penicillium*.

FRUIT ROT: LASIODIPLODIA THEOBROMAE (PAT.) GRIFFON ET MAUBLANC

Symptoms.—A somewhat dry rot of papaya fruit is due to the attacks of this fungus. The diseased fruits are characterized by the production of a sooty black mass of fungus spores on the surface.

Causal organism.—This fungus is the same as that producing a dry rot of cacao pods, root crops, and other vegetables.

Control.—All fruit rots may be controlled by taking care that no injuries are produced on the fruit during harvesting and that the fruit is used before becoming soft.

FRUIT ROT: PHYTOPHTHORA FABERI MAUBLANC

Symptoms.—This fungus may cause a soft rot of the mature fruit. The rot starts usually at some injury and spreads until the entire fruit becomes involved. Diseased fruits are covered by a white fungous growth.

Causal organism.—The organism producing this disease is the same as that producing the black rot of cacao pods. Conidia and oöspores are developed in abundance by the fungus. The fungus grows well in pure culture, being easily obtained by

simple plating out methods. It is more fully discussed under black rot of cacao pods.

Control.—The fruit should be handled so as to avoid injuries, and it should be used before it gets overripe.

LEAF ROT: MYCOSPHAERELLA CARICAE SYDOW

Symptoms.—This is a common leaf spot which, at times, may severely attack plants, causing a lack of vigor and a premature dropping of the older leaves. Circular spots, from a few millimeters to a centimeter in diameter, are produced. Older spots have an ashen-gray center surrounded by concentric light-brown rings bordered with darker brown. In the center of the older spots the minute black perithecia are produced.

Causal organism.—The perithecia are produced under the epidermal layer. They are more or less globular and brown with a distinct netted wall marking. An ostiolum is present at one end of the sack, protruding through the epidermal layer of the leaf (fig. 8). The asci, borne within, are elongated, club-shaped bodies containing typically eight two-celled, hyaline, vacuolated spores (fig. 8).

Control.—Since this disease is of minor importance, no specific control measure need be practiced. The collection and burning of all fallen or badly diseased leaves is beneficial in checking the fungus.

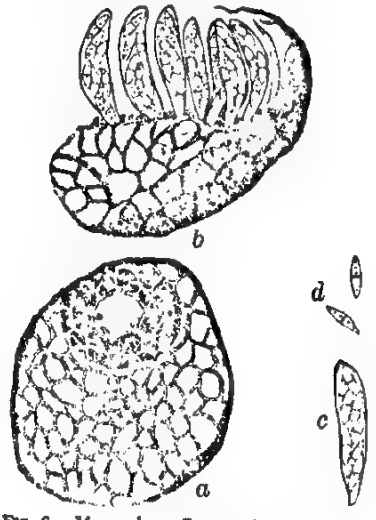

FIG. 8. *Mycosphaerella caricae* Syd. *a*, perithecium (× 326); *b*, broken perithecium showing production of asci (× 326); *c*, ascus with ascospores (× 326); *d*, ascospores (× 325).

POWDERY MILDEW: ERYSIPHACEAE

Symptoms.—Under favorable weather conditions papaya seedlings may be covered with a white powdery mildew. The disease is not severe.

Causal organism.—Typical erysiphaceous conidia and conidiophores are produced. No perfect stage of the fungus has been observed.

Control.—The disease is seldom severe enough to warrant a special control. Powdering plants with sulphur will check the disease.

Other fungi have been found on weakened and dead portions of the plant. *Aspergillus periconioides* Sacc. is commonly found on weakened and fallen leaves. *Colletotrichum papayae* (Henn.) Syd., *Diplodia caricae* Sacc., and *Didymella caricae* Tassi. have been found on dead and dying petioles. *Fusarium heveae* Henn. may be found on dead trunks.

CITRUS SPP. ORANGES, LEMONS, LIMES, POMELOS

Citrus culture is carried on in certain sections of the Islands. As is also true with the majority of the other fruit crops, little care is given citrus trees in the way of cleaning up, pruning, spraying, and cultivating. Consequently the trees are sickly, and in many cases they are severely attacked by insects and fungi.

BARK ROT

Symptoms.—Citrus trees growing in neglected and poorly kept orchards may be seriously attacked with a bark rot. The first indication of the rot is a slightly raised, sometimes cracked portion, from which usually a drop of gum oozes. These portions gradually increase in size; gum, in most cases, oozes out in more abundance; and in the latter stages a froth is present, indicating the presence of yeasts and other saprophytic organisms. These older cracked and rotted portions may be 0.5 to 5 centimeters wide and from 3 to 10 or 15 centimeters long, extending lengthwise with the trunk. In the older cases the bark gradually sloughs off, producing an irregular rotted portion in the bark down to the wood.

An internal symptom of new rots is a slight brownish watery discoloration. Usually there is a green coloration produced just below the rotted portion. This green coloration appears to be due to the abnormal production of chlorophyll. Older rotted portions may also show this greenish coloration, but the diseased parts are more or less brownish and usually covered with a watery frothy substance. A disagreeable odor is present in the older cases of disease.

Causal organism.—No work has been done with the causal organism. The disease appears to be produced by a definite organism.

Control.—Since the cause is not known no definite control can be assigned. Trees that are neglected and given poor culture seem to be more severely attacked. All badly diseased branches should be removed and burned. This with proper culture measures will reduce the disease to a considerable extent.

CANKER: PSEUDOMONAS CITRI HASSE

This infectious and destructive disease is widely prevalent in the Islands. On the commonly planted citrus fruit, *Citrus nobilis* Lour. (Satsuma orange, Canton mandarin), the disease is only slightly prevalent and does little damage. It is, however, severe on certain species in the college plantation at Los Baños, Laguna, where test varieties are grown. These different varieties are affected in the following order, the first-named being the most severely attacked: *Citrus maxima* (Burm.) Merr. (*Citrus decumana* Linn.) (large pomelo, bitter pomelo, djersek boli), *Citrus* sp. (Kusaie lime), *Citrus* (large orange), *Citrus* (Lisbon lemon), *Citrus* (Washington navel), *Citrus* (rough lemon), *Citrus medica* Linn. (citron), *Citrus nobilis* Lour. (Satsuma orange, Canton mandarin), *Citrus* sp. (small orange), *Citrus hystrix* DC. (wild lime), *Citrus mitis* Blanco (calamondin), and *Citrus japonica* Thunb.

This list is based on young plantings, and the order of attack will probably change somewhat as observations continue. A great variation occurs in the susceptibility of different varieties in the same species.

Citrus maxima (Burm.) Merr. (*Citrus decumana* Linn.) is most severely attacked when young. Older trees of native varieties grown by the Filipinos in the neighborhood of the college are attacked, but as with *Citrus nobilis* Lour. little damage is done.

Symptoms.—The characteristic appearance of the disease as it occurs on citrus is as follows:

Spotting is produced on leaves, stems, and fruit. At first the spots on leaves are small, round, watery, slightly raised dots. These dots enlarge, turn brown, extend through the leaf, become raised on one or both surfaces, and have ruptured surfaces. Concentric, irregular rings may be produced in the brown portion of the spots. A light yellow border is produced around the brown center. Frequently spots run together, producing an elevated, elongated, ruptured, brownish blotch (Plate III, fig. 2). In many cases a leaf-mining insect carries the infection through its winding gallery, or mine, in the form of a chain of canker spots (Plate III, fig. 3). This miner is the larva of a small moth, *Phyllocnistis citrella* Stainton, and is common throughout the Orient, being especially injurious to nursery stock.

On the twigs the spots are somewhat different. At first they are similar to those on the leaf, but later become irregular,

raised, spongy brown spots, often with a dark brown border. Spots are cankerous and persistent, but are only formed in the outer layers of the bark tissue. Frequently twigs are entirely encircled by cankers, but do not seem to be killed outright in all cases (Plate IV, figs. 1 and 2).

In the first stages of the disease the spots on the fruit are similar to those on the leaf. They may be scattered, but frequently run together, forming irregular, raised, brown cankerous blotches. The surface of the fruit is depressed or slightly wrinkled in the neighborhood of large blotches (Plate IV, figs. 2 and 3). Cankers do not penetrate deeply below the surface and seem to do little harm other than producing an unsightly appearance. Gumming of the fruit is associated with the disease in some cases, but this is not general.

Causal organism.—The bacteria causing this disease gain entrance to the host primarily through injuries. Citrus leaves, especially those of the highly spiny species, have many injuries due to the whipping of the leaves against the spines. The organism is spread throughout the tree primarily by rain. The bacteria grow well in pure culture, producing a yellow pigment.

Control.—The control of citrus canker is rather difficult. The orange, *Citrus nobilis* Lour., is the most commonly cultivated species in the Philippines and is relatively free from the disease; so no control measure need be applied for this species. Other species are, however, severely infected. For these control measures should be practiced. In order to obtain an effective control for citrus canker, persistent efforts must be used. This is true of the majority of bacterial diseases of fruit trees. A combination control of spraying and pruning out of the diseased portions will produce beneficial results. It is necessary, however, to be on the lookout for new infections, and these must immediately be removed. Monthly sprayings with Bordeaux mixture, to which a sticker has been added, is the most beneficial. Lime-sulphur must be applied in place of Bordeaux from time to time, in order to combat aphids and scale insects. In severe cases of infection it is advisable first to remove diseased leaves by spraying with a strong formalin spray (0.4 to 0.5 per cent), which will cause them to drop off.

CHLOROSIS: NONPARASITIC

Symptoms.—Frequently leaves on certain trees show a general yellowing in contrast to a definite mottling as produced in mottled leaf. In some cases this yellowing may cover the entire leaf, while in others large yellow blotches are produced. A

uniform yellowing of the leaves seems to be due to malnutrition, probably a lack of nitrogen. The yellowing in blotches may be due to the attacks of mites.

Control.—The disease may be avoided by using healthy stock and by the practice of proper culture methods. In case of insect attacks, these must be controlled by entomological methods.

DAMPING OFF: RHIZOCTONIA

Symptoms.—Seedlings grown in unsterilized soil and in poorly aërated places may be severely attacked, just at the ground surface, by this fungus, which first causes a browning of the stem and later a shrinking and weakening of the tissue, causing the plants to fall over and die. The disease is somewhat similar to, but more prevalent than, that produced by a *Sclerotium.*

Causal organism.—This organism is a common soil fungus causing a large amount of destruction to tender plants during periods favorable to its spread. It grows well in pure culture, first producing a coarse white mass of mycelium, which later turns brownish and produces a large number of brown sclerotial bodies. No spores have been observed. The fungus penetrates the plant tissues, causing the weakening and death of the cells.

Control.—Seedlings should be grown in sterilized soil and should be placed where there is plenty of chance for air.

DAMPING OFF: SCLEROTIUM

Symptoms.—Seedlings growing in damp and poorly aërated places are frequently attacked by a fungus that causes a rot resulting in damping off. The stem is attacked near the ground and becomes browned, shrunken, and weak, due to cell destruction. Plants in the latter stages of the disease fall over and die.

Causal organism.—Isolation and inoculation experiments show this disease to be due to a fungus that produces sclerotia. The fungus invades the tissues from the ground. Upon death of the plant small, round, smooth brown sclerotial bodies are produced. These bodies germinate directly by the production of mycelium. No spores have been observed. The same fungus may cause a damping off of coffee seedlings, cacao seedlings, and other plants. In pure culture a dense white growth is first produced, which later gives rise to a large number of round, smooth brown sclerotial bodies.

Control.—The disease is easily controlled by growing plants in well-aërated places, free from too great humidity. If the soil be heavily infected with the fungus, soil sterilization must be practiced.

DIE-BACK

Symptoms.—Die-back is common in poorly kept orchards and appears, in the main, to be due to a lack of nutrition. The symptoms are a gradual dying back of the branches, starting from the tip.

Causal organism.—No definite causal organism has been assigned. Many fungi are found on dead and dying twigs, including the following: On *Citrus nobilis* Lour.; *Zignoella nobilis* Rehm., *Cytospora aberrans* Sacc., *Eutypella citricola* Speg., *Hypoxylon atropurpureum* Fr. (on coccids), *Valsaria citri* Rehm., *Massarina raimundoi* Rehm., *Tryblidiella rufula* (Spreng.) Sacc., *Diaporthe citrincola* Rehm., *Diplodia aurantii* Catt., and *Tryblidiella mindanaensis* Henn; and on *Citrus maxima* (Burm.) Merr. (*Citrus decumana* Linn.); *Eutypella citricola* Speg. and *Eutypella heteracantha* Sacc. Growing on the latter fungus has been observed another fungus, *Nectria episphaeria* (Tode.) Fr.

Control.—Citrus culture in the Philippines is practiced in a slipshod manner. Die-back may be largely avoided by the use of correct culture methods. All dead and dying branches should be pruned out and burned.

EPIPHYTES: LORANTHUS PHILIPPENSIS CHAMISSO

Symptoms.—Epiphytes are sometimes found growing on trees in poorly kept plantations. They can be easily removed by pruning.

FRUIT ROT: LASIODIPLODIA THEOBROMAE (PAT.) GRIFFON ET MAUBLANC

Symptoms.—A dry rot of citrus fruit may take place due to the attacks of this common dry rot organism. Diseased fruits are characterized by a shriveled, dry appearance and are covered with a dense black sooty mass of spores.

Causal organism.—The organism gains entrance into the fruit through injuries. A series of pycnidia is produced just under the surface of the fruit, and from there, through openings extending to the surface, the spores are expelled in large numbers. The spores are, when immature, single-celled, hyaline, very granular, oval bodies. Upon reaching maturity, they become two-celled and dark brown. Germination takes place readily within a few hours in water. The spores may germinate before reaching the two-celled stage. The fungus grows well in pure culture, producing, on potato agar, a heavy growth of dark greenish to black mycelium. No spores have been observed in these cultures.

Control.—Care should be used in handling the fruit so as to keep it free from injuries.

FRUIT ROT: PENICILLIUM

Symptoms.—Fruit rots are present on fruit kept for some time out of storage. The *Penicillium* rot is characterized by the production of a green powdery mass of spores over the soft, rotted area. The rot starts at some injury and gradually spreads until the entire fruit is involved.

Causal organism.—The fungus penetrates the tissue of the fruit, causing a soft rot. It produces an abundance of typical *Penicillium* spores on the surface of the fruit. These spores blow from diseased to healthy fruit, thereby causing infection.

Control.—The fruit should be kept free from injuries. It should be used as soon as possible, and if stored it should be kept in a well-aërated place so as to avoid excessive moisture.

Phyllosticta circumsepta Sacc. has been found on the dying rind of fruit.

GUMMOSIS

Symptoms.—A gumming of the trunk, stem, and fruit occurs. Whether this is due to unfavorable climatic conditions, to lack of cultivation and care, or to parasites has not been fully determined. The disease of the stems is more severe in poorly kept orchards. Insect punctures in the fruit have been observed to result in a gumming; mechanical or fungus injuries, as in the case of citrus canker, may also cause a gumming. It appears that gummosis of stem and fruit here is not caused by any one definite organism or factor.

LICHENS

Symptoms.—Lichens are found in abundance, growing over all woody parts and even upon the leaves of trees, producing greenish gray blotches. The damage done appears to be slight; however, the normal physiological activities of the plant must be disturbed thereby.

Control.—Lichens can be reduced by the use of a spray or wash of 6 per cent copper sulphate solution or by judicious spraying, as discussed under citrus canker.

MOTTLED LEAF: NONPARASITIC

Symptoms.—Leaves thus diseased are characterized by a distinct yellowing of the leaf mesophyll between the large lateral veins. The tissue adjacent the midvein and the larger lateral veins is of a healthy green. Entire trees may be affected, but often only leaves on special branches are diseased. When the entire tree is affected, it is much dwarfed and may later die, due to secondary agencies. Badly diseased trees commonly show

witches'-broom effects and a more or less complete rosette of the leaves (Plate III, fig. 1).

Causal organism.—The disease is a nonparasitic one, being due to some disturbance of the normal physiological activities of the plant. It is not transmitted from one plant to another. Sometimes in marcotting, the disease is produced on branches the bases of which have been encircled with a bamboo tube or a coconut husk containing earth.

Control.—Since the disease is little understood, no definite control can be given. Badly affected trees should be removed, for they are stunted and will never produce healthy fruit.

PINK DISEASE: CORTICIUM SALMONICOLOR BERK. ET BROOME

Symptoms.—This disease may be severe during the rainy season in poorly kept orchards. The fungus is a common one, producing disease on other woody plants. Infection starts on the trunk or branches usually in some damp pocket. It is first noticed by the production of cracks and by an exudate of gum. As the fungus penetrates into the bark, it spreads under the surface and causes a more or less cankered condition. In the latter stages the bark cracks and dries up. The fungus may penetrate through the bark into the cambium and wood. When a branch or trunk of a small tree is girdled by the fungus and the xylem is invaded, the upper parts of the plant gradually die, due to starvation. Diseased trees are easily discovered by reason of the dead branches. The diseased area in certain stages of development, especially during the rainy season, is covered with a mass of pink mycelium that often extends over the bark in strands. During drier weather the mycelium dries considerably and is not so evident, as it changes to a dirty white or gray.

Causal organism.—No detailed work has been done with the fungus. It grows in pure culture, producing a matted mass of pinkish mycelium. The complete life cycle of the fungus has not been worked out.

Control.—Since healthy, vigorous trees are less liable to attack, proper cultural methods should be practiced. Spraying healthy trees as in the case of citrus canker will exclude the fungus. Once the fungus has gained entrance into and under the bark, spraying will do no good. Young infections may be removed by cutting out all the diseased portions well down into the healthy wood and painting the wound with a creosote paint or white lead. All badly diseased branches should be pruned out and destroyed by burning. These branches should be cut out 15 to 20 centimeters below the visible extent of the disease, for the mycelium

often penetrates farther than can be seen with the naked eye. All large wounds should be painted with a creosote paint or white lead. Severely infected trees should be cut down and burned immediately.

SCALY BARK

Symptoms.—A disease characterized by a scaling of the bark is common, but the causal factors have not been determined. The attacks of an insect just below the bark cause a sloughing, but this does not appear to be the only factor.

SOOTY MOLD: MELIOLA

Symptoms.—Frequently this sooty mold is found growing over leaves, stems, and fruit. It is superficial, growing on the sugary exudate of aphids. The fungus has been observed on *Citrus medica* Linn., *Citrus nobilis* Lour. and on *Citrus maxima* (Burm.) Merr. (*Citrus decumana* Linn.) and undoubtedly occurs on other *Citrus* species.

Causal organism.—A dense mass of brown mycelium, with its characteristic hyphopodia, is produced over the affected area. Setæ and dark brown spherical perithecia are produced from the mycelium (fig. 9). Within the perithecia are found the hyaline globular asci with from two to four typical, five-celled brown ascospores (fig. 9).

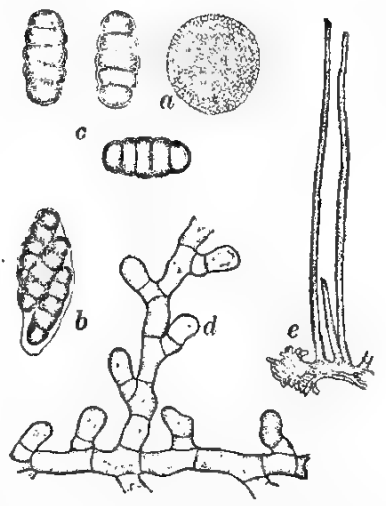

FIG. 9. *Meliola*, on *Citrus nobilis* Lour. *a*, perithecium (× 75); *b*, ascus (× 310); *c*, ascospores (× 310); *d*, mycelium with hyphopodia (× 310); *e*, setæ (× 75).

Control.—The disease may be controlled by spraying with lime-sulphur, which will keep the aphids under control as well as destroy the fungus.

SPINY MOLD: IMPERFECT FUNGUS

Symptoms.—A spiny mold may be produced on leaves, stems, and fruit. Black tufted masses of fungus appear in spots or frequently in masses, covering the entire affected portions. The fungus grows primarily on the exudate from aphids.

Causal organism.—A dense mass of brown mycelium, with numerous setæ, is produced. The setæ are septate and much elongated and they give the tufted appearance. Hyaline, elong-

ate, sometimes crescent-shaped granular spores are produced among the setæ. The fungus has not been identified.

Control.—The control is similar to that discussed under sooty mold of citrus.

WITHER TIP: COLLETOTRICHUM GLOEOSPORIOIDES PENZIG

Symptoms.—A gradual dying of twigs and branches is frequently produced by this fungus. Not only the twigs, but the leaves and the fruit may be infected. The leaves wither, and the twig is killed and shrinks, leaving a definite line of demarcation between healthy and diseased wood. On the leaf, dark brown spots are produced. The fruit beneath a withered tip branch often becomes infected, which is evidenced by a russet appearance. Minute black specks are produced over the diseased surface.

Causal organism.—The organism is evidenced by the acervuli, produced in the form of black specks over the diseased parts. The acervuli are formed under the surface, but later rupture it. Setæ are produced, and from a dense mass of short conidiophores are produced the minute, cylindrical, granular hyaline spores. The fungus grows well in pure culture, producing scanty mycelium, from which arise many small black fruiting bodies.

Control.—All diseased portions should be removed by pruning out well below the visible advance of the disease. Spraying with Bordeaux mixture as discussed under citrus canker is effective.

Gloeosporium intermedium Sacc. is found on injured citrus leaves, where it produces minute black specks in the gray injured portions. *Aschersonia sclerotoides* Henn. may be found growing parasitically on coccids that are on the leaves. A *Micropeltis* also may be found growing on leaves.

COCOS NUCIFERA LINN. COCONUT

BUD ROT: BACTERIAL

This is the most serious coconut disease in the Philippine Islands, if not in the world. Fortunately it is severe only in a few localities of the coconut regions, chiefly in Laguna, Batangas, and Tayabas Provinces. These coconut sections are some of the most extensive in the Islands and, unless control measures are carried out, the disease will spread.

Symptoms.—The first symptom is a withering of the youngest unfolded leaf, followed by the leaf's turning brown. Gradually other leaves wither and turn brown, until the entire central group is affected. At this stage the disease is easily recognized by the group of dead young leaves of the central bud, which has

become brown. Often some of the largest leaves of the bud fall over (Plate V, figs. 1, 2, and 3). This diseased central portion is surrounded by older leaves, on the outside, which are perfectly healthy and remain upon the tree until they drop off naturally. Trees are more commonly affected when they first come into bearing. The young nuts, on bearing trees attacked by the disease, remain small and fall off prematurely. Trees are affected most generally in regions of great moisture and in overcrowded areas.

Internal symptoms of diseased trees are very characteristic. A longitudinal section of the bud shows, in new cases, that the disease may start in the young leaves, at a point where they begin to unfold (Plate VI, fig. 1). At this point a spotting of the leaf is first noticed, then the organism works downward, causing a soft rot and browning of the group of unfolded leaves. The upper exposed portions of these leaves die and turn brown, due to the rotting beneath. The rot advances downward through the young leaves to the growing point and then spreads into the soft tissue below. From here it invades the woody tissue, usually not penetrating farther than from 5 to 10 centimeters. In the early stages of the disease no discoloration is produced in the growing point and cabbage, but a dark red to brownish ring always limits the advance of the disease in the wood on bottom and sides (Plate VI, fig. 4). The disease does not penetrate readily into the old leaf sheaths surrounding the young, tender, developing leaves (Plate VII, figs. 1, 2, and 3).

The rot is checked, as a rule, when it reaches the firmer tissues of the trunk, penetrating, in advanced cases, about 20 centimeters (Plate VI, figs. 2 and 3). The softness of the affected portion in the trunk is shown by the fact that the finger can be pushed into the diseased part. A vile, somewhat sour odor accompanies the disease. The most advanced stages of the disease are characterized by the white cabbage changing into a semiliquid mass with an ill-smelling odor. The diseased portion of the trunk becomes a mass of fibers and a semiliquid.

The disease spreads very rapidly from tree to tree, but the manner of spread is not fully understood. Insects are undoubtedly one of the factors to be considered in its transport from infected to healthy trees. In one barrio under observation, fifty-eight new infections appeared within one year after an inspection in which all trees found with the disease were cut down and burned. Infection must have started from one or a few trees unobserved during this first inspection. These trees are located in the upper extremity of the coconut region on the

slopes of Mount Banahao, where it is very damp. The trees are also planted too thickly. Both these factors are favorable to the development and spread of the disease.

Causal organism.—Microscopic examination of diseased tissues taken from typical young cases of bud rot showed no evidence of mycelium, but an abundance of bacteria. Diseased pieces collected under sterile conditions in the field and placed immediately into sterile vials developed no fungi; however, they were completely invaded with bacteria. Many fungi would develop from older diseased portions when placed in a moist chamber, but under no conditions was one specific organism always produced.

Careful inspection was made of over thirty typical cases of diseased trees. These trees were cut down and the bud opened for observation. In all cases the disease appeared to be due to bacteria. Isolations were made from sixteen different typical cases.

Cultures were obtained by cutting and plating out, under sterile conditions, small pieces from all parts of infected trees, from the tip of the unfolded infected leaves down to the growing point and into the wood below. Poured plates from these cultures showed that in the majority of cases a mixed culture of bacteria was present. In very young cases of infection, however, only one organism is present. The latter cases are hard to obtain, because saprophytic bacteria find a favorable place for development in the infected portion, and they are soon washed down into these parts. In order to prove the virulence of the bacteria isolated, a large series of inoculations was carried out.

These inoculations were made chiefly with seedling coconuts. The plants were from 60 to 180 centimeters tall. They were carefully prepared for inoculation by stripping off the outermost, older leaves. Then the portion to be inoculated was washed with mercuric bichloride, 1 to 1,000. With sterile scalpels, stabs were made into the growing point, and the pure cultures of bacteria were introduced. The injuries were then covered with paraffin. Over two hundred inoculations have already been carried out in this fashion and typical cases of bud rot produced (Plate VI, figs. 5 and 6). The first inoculations were not repeatedly positive, because they were made outside during the excessively dry season, under which condition the organism is not extremely virulent. In later inoculations made in a specially constructed damp chamber, the disease could be produced at will with the correct organism. By this method all the saprophytic bacteria were eliminated. Inoculations with fungi also proved negative.

After this eliminating process, there was left one distinct organism that would produce the disease. At least 75 per cent of positive infections can be obtained under proper conditions. This one organism has been carried through a series of three different plants by inoculation, reisolation, and reinoculation.

The organism produces white colonies with a bluish tinge. Since *Bacillus coli* (Escherich) has been associated with the disease in Cuba and since the organism isolated here in the Philippines appears to be somewhat similar to *Bacillus coli* (Escherich), inoculation experiments were carried out with the latter organism.

Authenticated cultures of *Bacillus coli* (Escherich) obtained from the United States and also cultures obtained from the Philippines were used. The cultures from the United States were isolated from man, those from the Philippines were isolated from man and horse. A bud rot was produced with each of these cultures. The rot produced from the first inoculation was very slight, but the organism reisolated and then reinoculated produced a rapid and severe case of rot. The initial inoculation was rather difficult to obtain, except in cases where the tissues of the coconuts were severely injured. This indicates that these bacteria must first pass through a weakened host before they become extremely virulent.

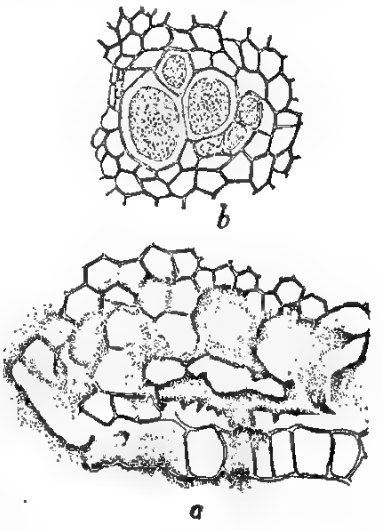

FIG. 10. Bud rot of coconut. *a*, cross section of infected portion of young unfolded leaf, showing mass of bacteria in tissue (× 425); *b*, cross section of infected portion of young unfolded leaf, showing mass of bacteria in xylem tubes of a vascular bundle (× 330).

As yet culture studies have not progressed far enough to assign a definite name to the organism isolated from coconuts here in the Philippines, but investigation has shown that there is a bacterium that causes the bud rot of coconuts. A complete and detailed acount of these investigations will be soon published.

Cytological studies show only the presence of bacteria. Sections from a typical case of bud rot were made from diseased portions obtained from the young leaves leading to the growing point, from portions of the growing point, from the cabbage, and in the wood. These sections show that the organism is not

only present in the parenchymatous tissue, but also that the chief means of spread in the plant is through the vascular system. Xylem tubes in the young leaves and in all portions down to the woody tissue are infected (fig. 10). This accounts for the rapid advance of the disease in the tissue.

Control.—Trees when once affected never recover. The mode of growth of the palms and the nature of the disease make it impossible to cure trees already infected. The only control so far determined is one of prevention of spread. All diseased trees should be cut down, and the diseased portions should be completely burned or deeply buried after sprinkling with lime. If this precaution of burning all infected trees be carried out under strict supervision, the danger of spread is largely eliminated.

The greatest factors in the severity of the disease are the growth of coconuts in excessively damp places and in extremely thick plantings. New plantings should be made only in those localities that are best suited for coconut growth and development. Plantings should not be too thick. The recognized distance for plantings for the best production and at the same time for the best control against bud rot is 10 meters each way.[2]

LEAF SPOT: EXOSPORIUM DURUM SACCARDO

Symptoms.—A spot that is not common and causes little damage. It is characterized by the production of black tubercular or wartlike bodies, the sporodochia, on the surface of leaves. These spots are scattered, sometimes densely, over the leaf surface. In some cases the sporodochia may be surrounded by a light yellowish discoloration of the leaf (Plate VIII, fig. 4).

Causal organism.—The wartlike bodies, or sporodochia, have no spines. The conidia are borne on conidiophores and are yellowish to brown and septate.

Control.—Since the disease is not severe, no special control need be practiced. All fallen diseased leaves should be collected and burned, so as to avoid a spreading or an epidemic.

LEAF SPOT: PESTALOZZIA PALMARUM COOKE ET GREVILLE

Symptoms.—This disease is common throughout all coconut regions. As a rule, it is not severe and causes little damage. The vitality of the tree is lowered, and in a few cases, especially on younger trees, the spotting may become severe. Spots often are scattered over the entire leaf surface. Young infections are characterized by small brown to black, elevated, circular spots a

[2] See Copeland, E. B., The Coco-nut. London, Macmillan and Co. (1914).

few millimeters in diameter. Older spots are irregular-circular to slightly oblong, may run together, and are from 1.5 centimeters to 2 or 3 centimeters long. These spots have a light brown to ashen-gray center and are bordered with a narrow dark brown ring (Plate VIII, fig. 3).

Causal organism.—In the gray parts are produced the characteristic minute black acervuli, which contain the spores. Spores are septate, with central brownish cells and hyaline end cells. Two to four hyaline appendages are produced at one end of the spores and usually only one at the other end (fig. 11). The fungus grows well in pure culture, producing, on potato agar, at first a felty mass of white mycelium, which later becomes studded with the black spore bodies. The agar in old cultures turns brownish.

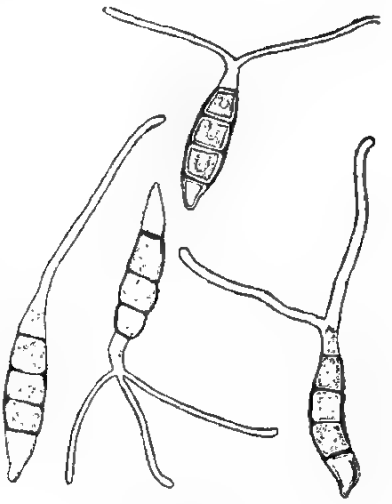

FIG. 11. *Pestalozzia palmarum* Cke. et Grev. Conidia, showing characteristic appendages (× 990); from pure culture.

Control.—In severe cases of infection of young trees, spraying with Bordeaux mixture is effective. Sanitation in the form of burning dead and diseased leaves is the usual control.

SOOTY MOLD: CAPNODIUM FOOTII BERKELEY ET DESMAZIÈRES

Symptoms.—A sooty mold is often developed on the under surface of the leaves. This is produced by the fungus growing on honey dew of coccids; this mold is not at all serious.

STERILITY OF NUTS

Symptoms.—Frequently nuts are found that are entirely composed of husk. No meat or shell is developed within the husk (Plate VIII, fig. 1). The disease is undoubtedly a nonparasitic one, being due to some abnormal physiological condition of the plant.

OTHER FUNGI

Other fungi found upon the coconut include the following: *Chaetosphaeria eximia* Sacc. and *Phyllosticta cocophylla* Pass. on dying leaves; *Anthostomella cocoina* Syd., *Diplodia epicocos* Cooke, and *Coprinus fimbriatus* B. et Br. on dead petioles; *Palawania cocos* Syd., *Hormodendron cladosporioides* (Fr.) Sacc.,

and *Coniosporium dendriticum* Sacc. on dead spathes; *Coprinus friesii* var. *obscurus* Pat. on dead sheaths; *Rosellinia cocoes* Henn. on dead peduncles; *Eutypella cocos* Ferd. et Winge., *Diplodia cococarpa* Sacc., *Diplodia epicocos* Cooke var. *minuscula* Sacc., *Diplodia cococarpa* var. *malaccensis* Tassi., *Cytospora palmicola* B. et C., and *Peroneutypella cocoes* Syd. on husks; *Elfvingia tornata* (Pers.) Murr. and *Ganoderma incrassatum* (Berk.) Bres. var. *substipitata* Bres. on dead trunks; and *Gloeoglossum glutinosum* (Per.) Durant. on base of living tree.

COFFEA SPP. COFFEE

DAMPING OFF: RHIZOCTONIA

Symptoms.—A damping off and stem rot of seedlings similar to that discussed under citrus is found on coffee. Diseased plants have browned stems, which shrink and cause the plant to fall (Plate XIII, fig. 1).

Causal organism.—The causal organism is the same as discussed under citrus stem rot.

Control.—Seedlings should be grown in sterilized soil and in well-aërated places.

DAMPING OFF: SCLEROTIUM

Symptoms.—Coffee seedlings are frequently attacked on the stem just at and above the ground by a *Sclerotium* that causes a damping off. Infected stems are blackened and somewhat shrunken. The fungus may also spread to the leaves, causing an advancing black rot. Spherical brown sclerotial bodies may be produced on infected portions. The disease is most severe during the rainy season and on seedlings kept in damp places. Young plants are killed by the attack.

Causal organism.—In pure culture the fungus produces numerous small, smooth, spherical brown sclerotial bodies. Infection experiments have proved the virulence of the fungus isolated, but as yet all attempts to produce spores have failed. This fungus is the same as that which may cause a stem rot and damping off of citrus seedlings.

Control.—The disease is only severe when plants are grown in poorly aërated places. Seedlings should be grown in sterilized soil and well-ventilated locations.

FOOT ROT

Symptoms.—A rot of the trunk of older coffee trees may take place at the surface of the ground. The entire trunk of the plant is girdled, resulting first in a yellowing of the leaves and then in a gradual wilting and death.

Causal organism.—No organism has as yet been associated with this disease. Consequently no definite control can be given.

LEAF SPOT: MICROPELTIS MUCOSA SYDOW

Symptoms.—A leaf spotting that is found on *Coffea excelsa* Cheval. and is only of slight importance. Minute, scalelike,

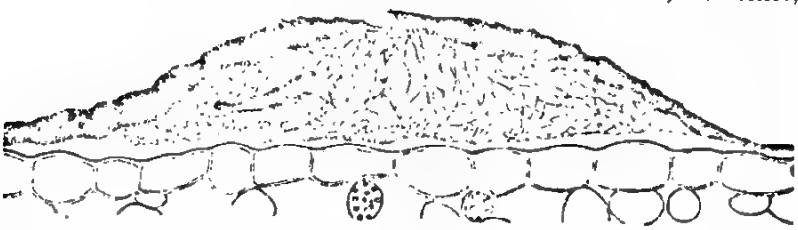

FIG. 12. *Micropeltis mucosa* Syd. Immature perithecium (× 335). The fungus does not penetrate leaf tissue.

raised black spots are scattered over the upper and lower leaf surface. They are usually more abundant on the lower surface.

Causal organism.—These scalelike black bodies are perithecia, within which are borne the asci and ascospores. The asci are clubshaped and contain six to eight hyaline three- or four-celled ascospores. The fungus is a superficial grower and does not penetrate into the leaf tissue (figs. 12 and 13).

Control.—The disease does little or no damage; consequently no control measures need be practiced.

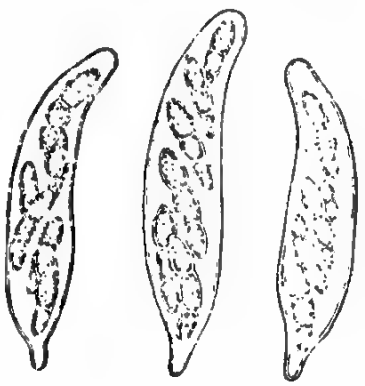

FIG. 13. *Micropeltis mucosa* Syd. Asci with ascospores (× 340).

RUST: HEMILEIA VASTATRIX BERKELEY ET BROOME

Symptoms.—This widely distributed and destructive disease has wiped out the coffee industry in various sections of the Islands. Circular or subcircular orange-red spots cover the under surface of leaves. Infected leaves wilt and drop, repeated attacks causing death to the entire plant. Young spots appear as transparent slightly yellowish discolorations. As the spot becomes older, the yellow increases, until finally a yellow dust, which turns to orange, is produced on the under surface of the leaves. The disease is most severe and evident during the rainy season.

Coffea arabica Linn., the best commercial coffee in this section,

is severely attacked by the rust and has been practically wiped out in most regions. A few favorably situated districts, in high altitudes, still produce Arabian coffee successfully. Owing to *Hemileia, Coffea arabica* Linn. is now of relatively slight importance in Java. Many plantations have been uprooted and replanted to *C. robusta* or hybrid varieties.

Causal organism.—The orange dust on the under surface of leaves is made up of the single-celled irregular uredospores and few single-celled teleutospores. The uredospores are irregularly obovate, bilateral, with short, blunt spines on the dorsal surface and with the ventral side smooth. They are produced on the leaf surface from stalks projecting through the stomata (fig. 14). The uredospores germinate readily in water. Penetration takes place by way of the stomata. The mycelium grows in abundance in the air spaces and in the intercellular spaces of the leaf tissue. Teleutospores are not produced in abundance. They are small, pale yellow, and smooth and have a short, slender, hyaline pedicel. They germinate often on fallen leaves by the production of a promycelium with sporidia (fig. 14).

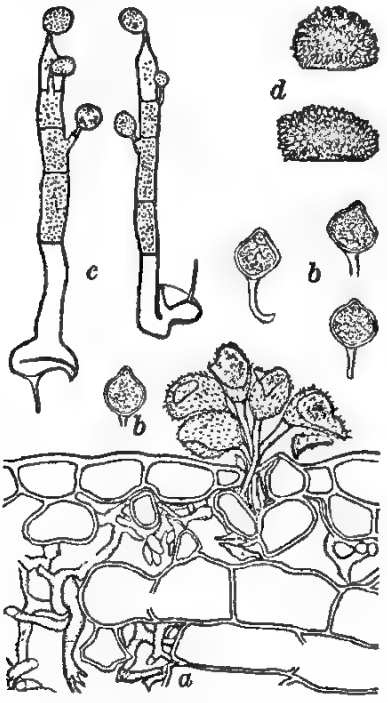

FIG. 14. *Hemileia vastatrix* B. et Br. *a*, infected coffee leaf, showing mycelium in tissue and production of uredospores some of which were cut in sectioning (× 325); *b*, teleutospores (× 325); *c*, germinating teleutospores, promycelia, and sporidia (× 325); *d*, uredospores (× 325).

Control.—Control consists in selecting resistant varieties and in spraying with Bordeaux mixture. As yet no resistant strain of *Coffea arabica* Linn. has been developed. In the Philippines, as shown by the College of Agriculture plantings, *Coffea robusta* is only slightly attacked and *Coffea arabica* Linn. is severely attacked. The *liberica* varieties need a special pulper, and the *robusta* coffee is of relatively poor quality and commands a lower price. The *arabica* coffee, *Coffea arabica* Linn., is the most easily handled and is very productive;

therefore, for the Philippines, the best control measure for this variety is spraying. Spraying experiments have shown that the disease can be controlled with Bordeaux mixture at a cost of 10 centavos [3] a tree per year.

SOOTY MOLD: AITHALODERMA LONGISETUM SYDOW

Symptoms.—A black sooty mold may be produced over the surface of leaves. Little injury is done, as the organism is not abundant.

STEM DISEASE

No important stem diseases on older plants have been observed. *Coniothyrium coffeae* Henn. has been found on twigs of *Coffea arabica* Linn.

COLOCASIA ESCULENTUM SCHOTT (COLOCASIA ANTIQUORUM SCHOTT). GABI

BLIGHT: PHYTOPHTHORA COLOCASIAE RACIBORSKI

Symptoms.—Gabi, which is extensively grown in the Philippines, suffers severely from the attacks of this fungus. Leaf blade, petiole, and corms are attacked. Leaf spots appear at first as small, roundish dark brown spots. They rapidly increase in size, may be circular, oval, or often running together, until finally the entire leaf is diseased. Spots are not confined to the portion of the leaves between main veins, but readily cross the latter. Spots 2 to 3 centimeters in diameter are dark and rather watery and produce drops of a yellow liquid. Older and larger spots have yellowish brown centers bordered by broad watery rings. Frequently the margins have concentric brown or yellow rings (Plate XV, fig. 1). When spots coalesce, covering the entire leaf, a soft, watery, disintegrating leaf is produced.

In severe cases the petioles may become infected. The fungus gradually invades the petiole, which becomes blackened, shrunken, and watery and finally collapses. The entire diseased leaf then decomposes into a watery mass.

Infection of the corm may occur in severe attacks and during damp weather. Diseased corms disintegrate with a wet rot. The disease is most severe during the rainy season.

Causal organism.—A downy mass of spores is not produced on the diseased spots, but only a delicate white growth can be detected. This is made up of the conidia produced on short

[3] One peso Philippine currency equals 100 centavos, equals 50 cents United States currency.

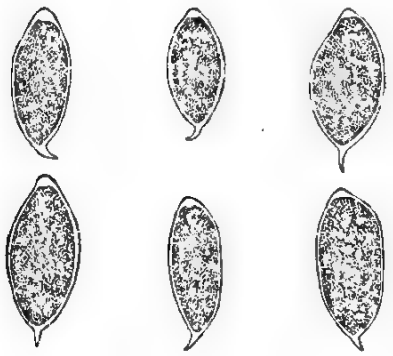

FIG. 15. *Phytophthora colocasiae* Rac. Conidia (× 330).

conidiophores. The conidia are large, thin-walled, smooth, and colorless and have short, broad papillæ (fig. 15). No oöspores have been observed. Infection takes place by the conidia, which are scattered chiefly by water.

The fungus grows readily in pure culture and can be easily isolated by the simple method of plating out diseased portions on potato agar. A downy mass of white mycelium develops on potato agar slopes, and conidia are formed in abundance. Sexual spores are produced in pure culture.

Inoculation experiments, in a damp chamber, produce typical leaf spots in two to three days.

Control.—Control consists in the growing of disease-resistant varieties. Spraying with Bordeaux mixture is effective.

Xanthosoma sagittifolium Schott, a heavy-yielding yautia, is not attacked by the *Phytophthora* and should replace the ordinary gabis.

CUCUMIS SATIVUS LINN. CUCUMBERS

DOWNY MILDEW: PLASMOPARA CUBENSIS (B. ET C.) HUMPHREY

Symptoms.—Yellow spots are at first produced on leaves. The whole leaf then turns yellow, shrivels, and soon dies. Central parts of older spots become dead and brittle and are a light brown. The disease starts with the older leaves and advances to the younger ones. Few cucumbers are produced on diseased plants.

Causal organism.—The typical branched conidiophores are produced singly or in small clusters from the stomata. Conidia are oval and light brown to violet-tinted.

Control.—Spraying with Bordeaux mixture should be done in severe cases of infection.

LEAF SPOT: CERCOSPORA

Symptoms.—Irregular to angular light greenish leaf spottings are found upon cucumbers. The spotting is not severe.

CUCURBITA MAXIMA DUCH. CALABAZA, SQUASH

DOWNY MILDEW: PLASMOPARA CUBENSIS (B. ET C.) HUMPHREY

Symptoms.—This disease is similar to that discussed under *Cucumis sativus* Linn.

POWDERY MILDEW: ERYSIPHACEAE

Symptoms.—A white powdery mass may be produced on the leaves. The disease is similar to that discussed under papaya and tomato.

Causal organism.—Typical conidia and conidiophores of the Erysiphaceae are produced.

Control.—Powdering with sulphur in severe cases of infection will check the disease.

DAUCUS CAROTA LINN. CARROT

STEM ROT: RHIZOCTONIA

Symptoms.—During damp weather a stem rot of the carrot may be abundant. The stems are attacked just at and above the ground. Infected stems become brown, shrivel up, and cause the death of the leaf by cutting off the water supply.

Causal organism.—Isolations and pure culture work showed the causal organism to be a *Rhizoctonia.*

Control.—Avoid planting during the excessively rainy season.

DIOSCOREA ESCULENTA (LOUR.) BURKILL. YAMS

LEAF SPOT: CERCOSPORA UBI RACIB., CERCOSPORA PACHYDERMA SYDOW

Symptoms.—Leaves may be moderately spotted with spots of the *Cercospora* type. Little injury is done.

LEAF SPOT: ELLISIODOTHIS REHMIANA THEISS ET SYDOW (PHYLLACHORA DIOSCOREAE SCHWEIN, PHYLLACHORA REHMIANA THEISS ET SYDOW)

Symptoms.—Shiny black stroma are scattered over infected leaves. Little damage is done.

RUST: UREDO DIOSCOREAE (BERK. ET BRM.) PETCH., UREDO DIOSCOREAE-ALATAE RACIBORSKI

Symptoms.—A common leaf trouble, which at times is serious. Characteristic yellowish rust pustules are developed on the under surface of leaves.

STORAGE ROTS: LASIODIPLODIA THEOBROMAE (PAT.) GRIFFON ET MAUBLANC

Symptoms.—Storage rots are present in abundance. This rot is characterized by the production of a sooty black mass of spores on the surface of dry-rotted roots.

Causal organism.—The organism causes a dry rot of a large number of root crops. It is more fully discussed under dry rot of cacao.

Control.—Avoid injuries in digging. Store in a well-aërated place. All diseased roots should be sorted out and burned.

A *Rhizopus* may also cause a rot.

Phoma oleracea Sacc., *Gloeosporium macrophomoides* Sacc., and *Phomopsis dioscoreae* Sacc. are found on dead stems.

Phyllosticta graffiana Sacc. and *Mycosphaerella dioscoreicola* Syd. are found on leaves of *Dioscorea esculenta* (Lour.) Burkill.

DOLICHOS LABLAB LINN. LABLAB BEAN

LEAF SPOT: CERCOSPORA

Symptoms.—Round gray-centered spots with purplish borders may be scattered over the surface of leaves. Little damage is done.

Causal organism.—Typical, elongate, septate, tapering *Cercospora* spores are produced on light brown conidiophores. The latter are formed in groups from the stomata.

Control.—Crop rotation will reduce the prevalence of the disease.

ORANGE GALLS: WORONINELLA DOLICHI (CKE.) SYDOW

Symptoms.—This disease is similar to that discussed under *Psophocarpus tetragonolobus* DC.

Septoria lablabis Henn. and *Septoria lablabina* Sacc. may be produced on weakened mature leaves. *Diplodia lablab* Sacc. is produced on the stems.

On dead Kultha beans, *Dolichos uniflorus* Lam., may be found the following: *Vermicularia horridula* Sacc. and *Didymella lussoniensis* Sacc.

FICUS CARICA LINN. FIG

RUST: KUEHNEOLA FICI (CAST.) BUTL. (UREDO FICI CAST.)

Symptoms.—A disease that may be very severe, causing defoliation, especially during the rainy season. Raised brownish sori are produced on the under surface of the leaf. Often the under surface is covered with a rusty powder composed of spores. Small yellowish spots are produced on the upper surface of the leaf above each sorus on the under surface.

Causal organism.—Usually cushion-shaped, light brown, spiny uredospores only are produced. Teleutospores are smooth, in chains, and with the germ pores apical.

Figs are not grown commercially in the Philippine Islands. Wild figs, of which there are many species in the Islands, have the leaves commonly spotted with the characteristic stromata produced by the genus *Phyllachora.*

GLYCINE MAX (LINN.) MERR. (GLYCINE HISPIDA MAXIM.). SOY BEAN, SOJA

BLACK MILDEW: TROTTERIA VENTURIOIDES SACCARDO

Symptoms.—Frequently entire patches of soy beans appear yellowish and sickly. This may be due to a fungus that makes

itself evident by the production of numerous, small black specks on the under surface of the leaves. Serious damage may be produced.

Causal organism.—The pycnidia are brown, with conspicuous wall markings, and they bear spines. Conidia are elongate, somewhat tapering, often curved, five- to seven-celled, and hyaline.

Control.—Crop rotation should be practiced.

BLIGHT: RHIZOCTONIA

Symptoms.—During the rainy season entire fields may be wiped out, due to this common soil fungus (Plate IX, fig. 1). The disease is most severe in close plantings. Soy beans are not the only plants attacked. All other beans and apparently every plant growing in a matted condition may be attacked. Aside from being found on beans, the disease has been observed on African peanuts, *Voandzeia subterranea* Thou., and on weeds growing among infected plants. Beans and other plants that can be grown on trellises, so as to keep them off the ground, and plants grown where they are not crowded, thereby permitting of sufficient aëration, are less subject to the disease.

Stems, leaves, and pods are all severely affected. The disease starts from the ground, growing up the older hardy stem to the tender portions or attacking the tender portions directly if they touch the ground. The mycelium of the fungus can be easily seen growing over the plants in a whitish mass and spreading from plant to plant. Infected leaves are at first somewhat yellowed in blotches, and gradually they turn black and disintegrate into a soft mass. Diseased plants touching healthy plants will afford a means of spread. From infected leaves the disease spreads to the tender stems and even to the more mature stems, causing them to decay and to turn into a watery mass. As the leaves and stems disintegrate, and especially during drier weather, countless numbers of sclerotial bodies are produced (Plate IX, fig. 3). These sclerotial bodies at first are white and soft, but soon turn brown and hard. They are sometimes roughly spherical, from 1 to 3 millimeters in diameter, or they may be somewhat flattened and elongated, often 6 millimeters in length (Plate X, fig. 3). The diseased leaves and sclerotial bodies fall to the ground, whence the latter produce mycelia during favorable weather and attack plants as before described. The disease is not severe during the dry season nor during the drier weather in the rainy season. It spreads with remarkable rapidity during damp weather.

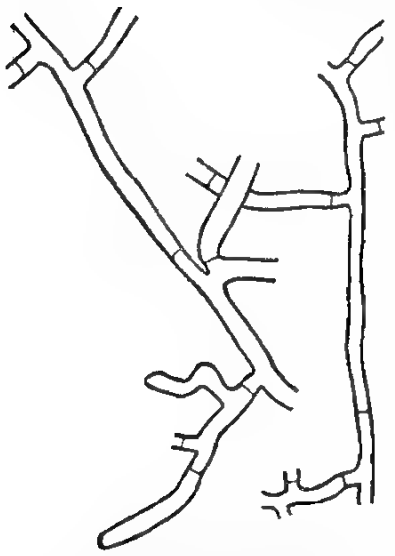

FIG. 16 *Rhizoctonia*. Mycelium from pure culture of fungus (× 340), isolated from *Glycine max* (Linn.) Merr. (*G. hispida* Maxim.). Note characteristic branching.

diseased plants. The advance of the fungus can be retarded or completely stopped by removing the bell jars and putting the plants in the sun. Reisolation from infected plants produced the same fungus used for inoculation. At no time in diseased fields or on pure cultures have spores been observed. Attempts to produce spore-bearing bodies and spores from sclerotial bodies have thus far failed. The mycelium is typical of *Rhizoctonia* (figs. 16 and 17).

In the cross inoculations cultures obtained from *Glycine max* (Linn.) Merr. (*Glycine hispida* Maxim.), *Voandzeia subterranea* Thou., and *Phaseo-*

Causal organism.—The fungus mycelium penetrates to all diseased portions, undoubtedly producing an enzyme, which aids in disintegration. Numerous inoculation experiments have been carried on, using different beans as hosts. Sclerotial bodies from pure cultures were merely placed on leaves or tender stems, and the plants were put under bell jars. Within two days infection and blight were produced. Sclerotial bodies produce mycelia direct and infect injured or uninjured portions. Within one week the entire plant is blighted and falls over in a soft mass (Plate IX, figs. 2 and 3). Later sclerotial bodies are formed on these

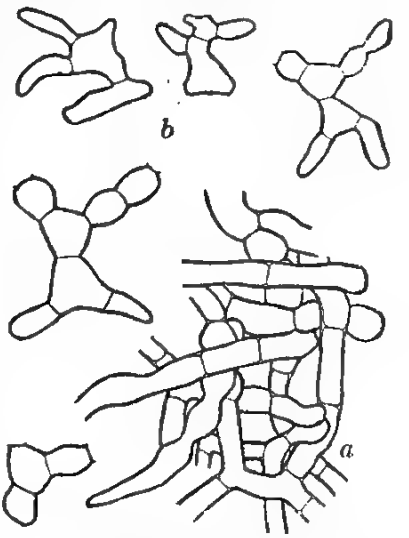

FIG. 17. *Rhizoctonia*. Mycelium from sclerotial body, growing in pure culture (× 340); *a*, formation of sclerotial body; *b*, portions of sclerotial body. Isolated from *Glycine max* (Linn.) Merr. (*G. hispida* Maxim.).

lus calcaratus Roxb. all produced typical disease on *Phaseolus lunatus* Linn., *Phaseolus vulgaris* Linn., and *Phaseolus calcaratus* Roxb., which shows that the organism causes a general blight of beans under suitable conditions.

Further inoculation experiments show that under suitable conditions this organism may attack and kill a large number of succulent plants (Plate X, fig. 1). A pure culture isolated from soy beans killed the following seedlings in an experiment carried out in a damp chamber: *Glycine max* (Linn.) Merr. (*Glycine hispida* Maxim.), *Voandzeia subterranea* Thou., *Zea mays* Linn., *Capsicum* spp., *Carica papaya* Linn., *Citrus maxima* (Burm.) Merr. (*Citrus decumana* Linn.), *Coffea arabica* Linn., *Anona squamosa* Linn., *Hibiscus sabdariffa* Linn., *Nicotiana tabacum* Linn., *Saccharum officinarum* Linn., and the woody seedlings *Passiflora quadrangularis* Linn., *Lonchocarpus* sp., and *Caesalpinia sappan* Linn. Seedlings only slightly attacked were *Eugenia uniflora* Linn. and *Tamarindus indica* Linn.

A coarse, dense mass of whitish mycelium is at first produced in pure culture. Later whitish bodies of mycelium develop, which enlarge and become hard brown sclerotial masses. The sclerotial bodies are connected by fibrils.

Control.—Since the disease is only severe during excessively damp weather, in thick planting and where plants form a mat over the ground, control consists in avoiding these conditions. Planting should be done so as to escape the heavy rainy season. Inasmuch as sclerotial bodies fall to the ground and remain alive for a long period, crop rotation will have to be practiced. In this crop rotation plants should be grown that do not form a mat over the ground. Care should be taken that no sclerotial bodies are sown with the seed.

DOWNY MILDEW: PERONOSPORA

Symptoms.—Light green blotches may be produced on the leaves. These spots are due to the destruction of the chlorophyll by the presence of the fungus. Young leaves are often wrinkled because of the more rapid growth of the cells about the points of infection. A light purplish to white downy growth is produced on the under surface of diseased leaves.

Causal organism.—This purplish growth is made up of large numbers of much-branched conidiophores at the tips of which the spores are produced. The conidia are somewhat ovoid and hyaline (fig. 18).

Control.—Crop rotation should be practiced.

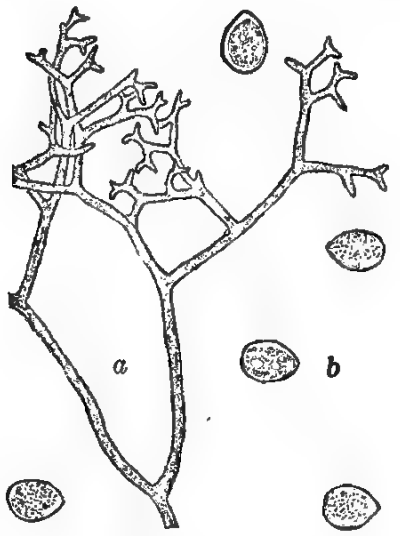

FIG. 18. *Peronospora*, on *Glycine max* (Linn.) Merr. (*G. hispida* Maxim.). *a*, portion of typical branched conidiophore (× 320); *b*, conidia (× 320).

RUST: UROMYCES SOJAE SYDOW

Symptoms.—Frequently soy beans may be severely attacked by this rust fungus. Characteristic brown rust sori are scattered thickly on the under surface of leaves. Spots are at first circular, raised brown blisters, but later burst open, exposing the spores. The upper surface of diseased leaves is yellowed above the sori on the lower surface.

Causal organism.—Irregular, short, spiny brown uredospores are produced in the rust sori (fig. 19).

Control.—Crop rotation should be practiced.

GOSSYPIUM SPP. COTTON

ANGULAR LEAF SPOT: BACTERIUM MALVACEARUM ERW. SMITH

Symptoms.—The disease is present on leaf, stem, and fruit. On the leaf the characteristic spots are from 1 to 4 millimeters in diameter; they are angular, with brownish centers bordered with light brown to yellow. Young spots are smaller and have a water-soaked appearance. They can be more easily detected on the lower surfaces of the leaves. Spots may run together forming brownish blotches which later become brittle. The dead brown tissue may fall out of the spots. Badly attacked leaves wither, die, and fall to the ground. The disease may be evident on the tender stalks in the form of blackened cankerous patches. On the bolls, at first, minute water-soaked spots are produced, which later may run together, producing sunken brownish or reddish brown blotches. If the bolls are young when attacked, the contents may be consumed; but on older bolls only the outer layers are invaded, producing little injury to the fiber. Young seedlings may be attacked first on the leaf from where the

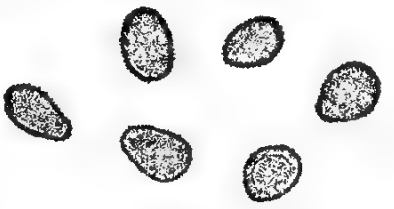

FIG. 19. *Uromyces sojae* Syd. Uredospores (× 315).

disease may spread to the stem, causing a blackened, water-soaked, weakened stem which finally falls over. In some cases, on older seedlings, only blackened blotches are produced. These may run together and girdle the stem, resulting in the falling over of the seedling.

Causal organism.—The causal organism is a bacterium that produces a yellow pigment in pure culture. It gains entrance into the plant through stomata and injuries. The organism may live over on the seed and lint for at least four months. It may also live in the soil for a considerable period.

Control.—The chief control consists in killing the organism on the seeds before planting. The seeds should first be delinted in sulphuric acid and then treated in hot water at 72° C. for eighteen minutes. In severe cases of plant infection, spraying with Bordeaux mixture will reduce the number of infected plants.

RUST: KUEHNEOLA DESMIUM (B. ET BR.) SYDOW [UREDO DESMIUM (BERK. ET BR.) PETCH]

Symptoms.—A common leaf rust found at the College of Agriculture on *Gossypium herbaceum* Linn. and on *Gossypium brasiliense* Macfad. Infected leaves are entirely covered on both surfaces with the minute brownish to black pustules. Little damage is done.

HEVEA BRASILIENSIS (HBK.) MUELL.-ARG. PARA RUBBER

BLACK ROT OF FRUITS: PHYTOPHTHORA FABERI MAUBLANC

Symptoms.—Diseased fruits are blackened, with a more or less watery discoloration, and rot upon the tree. The outer layer of the fruit shrivels, splits and dries up without maturing the seeds. Older diseased pods with matured seeds are shrivelled so that the seeds cannot be liberated. The disease is most severe during excessively damp periods and may cause the loss of the entire fruit crop. The fungus often grows from diseased fruits into the twigs causing a die-back. Usually the disease does not advance far down the twig. Diseased fruits serve as a source of infection for the stem canker.

Causal organism.—The causal organism is the same as discussed under *Hevea* and cacao canker and the black rot of cacao pods.

Control.—All diseased fruits should be collected and burned. Proper distances for planting and the sanitary precautions as discussed under the canker of Para rubber serve equally well in reducing the black rot of the fruits.

CANKER: PHYTOPHTHORA FABERI MAUBLANC

Symptoms.—The canker of Para rubber may be rather hard to detect in its early stages. In the Philippines the disease is similar to that discussed by Petch in Ceylon. External symptoms usually consist in a darkening of the bark, and in older cases there may be a definite demarcation of the diseased area. Most frequently the diseased area is smooth, but it may be cracked and scaly. During damp weather a reddish or purplish liquid sometimes exudes from the larger diseased areas. On older trees the disease cannot always be noticed from outward appearances, for a true cankered condition may not be produced. Internal symptoms are then the only indications of disease. Diseased trees cease to yield latex. The cortex, instead of its healthy white, yellowish, or clear red appearance, is characterized by a black layer produced under the outer brown bark and underneath this the cortex is discolored, in young cases gray, and in older cases a purplish red. In young cases only the outer layer of the bark may be diseased. This can be detected by carefully scraping the areas that do not produce latex to determine whether the cortex is blackish instead of being a healthy color.

When diseased trees have been cut down and piled ready for burning, they may be attacked by *Megalonectria pseudotrichia* (Schw.) Speg., which is characterized by a dense reddish mass of raised bodies, the perithecia, produced on the surface of the trees. This fungus is regarded as a saprophyte and is only found on the dead or weakened portions of trees. It may, however, gain entrance into diseased areas of living trees, consequently it should be guarded against.

Causal organism.—The Para rubber canker is produced by the same fungus that produces the black rot of *Hevea* fruits and also the black rot of pods and canker of cacao. The organism is more fully discussed under cacao. On rubber, so far as has been observed, only the conidial or sporangial stage is produced. Generally the asexual spore bodies are roundish or egg-shaped. Conidia germinate directly by the production of a germ tube that develops into the mycelium. These same spores under favorable damp or rainy conditions may germinate by the production of zoöspores. The spore body is then called a sporangium or a zoösporangium. The zoöspores swim about for a time, then come to rest and germinate as ordinary conidia by the production of a germ tube, which penetrates into the host primarily through injuries. The mycelium is almost always internal, spreading through the bark and is seldom found growing

over the surface. The fungus grows well in pure culture, producing on sterile potato cylinders, a dense white mycelium with conidia, sporangia, and chlamydospores.

Control.—All diseased portions should be carefully cut out, down to the healthy tissue, and burned. Disinfection of the knives used for cutting with a 2 per cent formalin solution is recommended. A careful inspection of the plantation should be kept up so that the cankers can be cut out when they first appear. All wounds made by cutting out the diseased cortex should be painted with a coal-tar preparation, care being taken not to paint the cambium layer at the edges of the cut surface. Cacao should never be planted with or near Hevea rubber. In severe cases of the disease it might be advisable to spray the trunks of young trees with Bordeaux mixture. This cannot be done with tapping trees. The humidity of the plantation should be lessened by admitting air and sunlight through the removal of intercrops, thinning out by pruning and planting according to the regulation distance, which will permit a ready aëration. All diseased trees and rubber trash should be burned as soon as possible to avoid the spread of *Phytophthora* spores. It might be advisable to obtain a large blast torch for this purpose.

LEAF SPOT: HELMINTHOSPORIUM HEVEAE PETCH

Symptoms.—Leaves of nursery plants a meter or more high may become spotted, but no serious damage has been observed. The spots may be thickly scattered over the leaf surface. When young they are minute, having purple centers with a lighter purple haze about the edges; older spots are circular, 3 to 5 millimeters in diameter, with white semitransparent centers bordered with a purplish ring. The disease has been observed on seedling plants only.

Causal organism.—The spores are produced on both surfaces of the leaf, but are more abundant on the lower surface. They are cymbiform, brown, and from eight to eleven septate. The conidiophores are scattered, simple, brownish, and septate.

Control.—Since the disease is not serious and never has been observed to cause defoliation, no control has been found necessary. If severe cases of infection should arise, spraying with Bordeaux mixture would control the disease.

PHYSIOLOGICAL TROUBLE

Symptoms.—This diseased condition is sometimes spoken of as brown bast. The external appearance of such trees is usually normal. Internal characters may be normal, but fre-

quently a gray to dark brown discoloration appears in the vicinity of the bast. The chief internal symptom is the stoppage of latex flow, due to some abnormal condition of the latex tubes.

Causal organism.—No causal organism has been associated with the disease. It appears to be due to some abnormal physiological condition, which may be inherent in certain trees; however, in certain cases trees appear to recover.

Control.—Tapping should be discontinued for a period of years on infected trees. Seeds for propagation should never be selected from diseased trees.

ROOT DISEASE: FOMES LIGNOSUS (KL.) BRESADOLA

Symptoms.—The disease is most severe upon young trees from 1 to 3 years old. Frequently diseased patches are produced in plantations. Diseased trees at first show a yellowing of the leaves, which is followed by a wilting and death. Dead trees can be easily pulled up or pushed over. The diseased roots are characteristically covered with a white mycelium, which may be in the form of strands spreading over the root or in the form of a sheet covering the entire surface. The white strands of mycelium spreading over the roots are the characteristic symptoms. These strands may be 0.5 to 1 centimeter broad and may be divided into finer strands that spread to the lower portion of the trunk and to the extremities of the roots. The diseased roots and lower trunk are not discolored, but become soft, like punk. The fungus also develops well on a number of jungle trees and stumps where it produces the same symptoms.

Causal organism.—The mycelium growing over the surface of the roots penetrates into the tissues, thereby causing death. The cortex and wood are completely invaded by the mycelium. From diseased roots the mycelium can spread through the ground to the roots of healthy trees. This is one of the chief methods of spread and accounts for the disease appearing in patches throughout the plantation. Fruiting bodies of the fungus are not usually produced on rubber trees, because the diseased trees are usually burned as soon as found. If diseased stumps are left standing, the characteristic fruiting bodies will be produced. They are more commonly found on stumps of jungle trees and are always produced above ground.

The fruiting bodies are at first orange yellow cushions, which later develop into flat, somewhat semicircular plates. They are usually 8 centimeters long, 4 centimeters wide, and 1 centimeter thick behind, but may attain a width of 30 centimeters. They are perennial and woody, belonging to the "bracket fungi." At first

the upper surface is red-brown with concentric dark brown lines. It is smooth with concentric grooves parallel to the outer edge. The lower surface is covered with minute pores, the spore-bearing surfaces, and at first is orange; but later, when old, is red-brown.

Control.—The disease as a rule cannot be detected until the tree is about to die; consequently remedial measures must be practiced that will prevent the fungus attack. Land cleared for rubber plantations should have the old jungle stumps removed and burned as completely as possible down to a depth of at least half a meter. Preferably the land should be cleared, cleaned, and planted to a cultivated crop two years before planting the rubber. This will give time for the complete removal and burning of all stumps.

Dead rubber trees must be dug up with all roots and burned. Since the disease frequently occurs in patches, these patches may be isolated by digging a trench, about 45 centimeters deep, around the affected trees. Quicklime should be scattered over the ground and in the trench. This will prevent the fungus from spreading through the ground to healthy surrounding trees. All dead stumps should be removed and the infected spot dug up so as to destroy as many of the roots as possible. Frequently newly infected trees near affected spots can be saved by removing all dirt from the tap roots and cutting out the affected portions. If the roots are too severely diseased, the tree must be dug up and burned. It is absolutely necessary to remove all dead stumps so as to prevent the spread of the disease by the mycelium growing through the ground, and to prevent the production of fruiting bodies, which produce spores that spread the disease. An efficient drainage system should be provided for poorly drained regions.

SPOTTING OF PREPARED PLANTATION RUBBER: SAPROPHYTIC FUNGI

Symptoms.—Prepared plantation rubber when produced under improper conditions may, during drying, become spotted with bright red, pink, reddish yellow, dark blue, bluish green, bright yellow, black, or clear spots. The colors can be more easily observed by holding the sheets of rubber up to the light. These spots may extend through the entire sheet, or they may be confined to the upper or the lower surface. They range from mere specks, 1 to 2 millimeters in diameter, to blotches, 15 centimeters in width. When the spots are abundant, a mottling of red or yellow may be produced. The color usually fades slightly after several weeks, but it has been observed to last for an indefinite period.

Causal organism.—The organisms causing the trouble in the Philippines have not been studied. In the Federated Malay States the following common saprophytic fungi have been assigned as the cause: *Penicillium maculans* sp. n., *Fusarium*, *Chromosporium crustaceum* sp. n., *Trichoderma koningi* (Oud.) Oudemans et Koning, *Eurotium candidum* Speg., and *Bacillus prodigiosus* (Ehrenb.) Fluegge. Oil and dirt are other sources of discoloration. The latex becomes primarily infected in the field due to improper field cultural methods, the use of contaminated water for washing out jars, and to contaminated pails.

Control.—Ordinary sanitary measures are sufficient for control. General cleanliness in tapping, collecting of latex, and preparation of rubber should be observed. The plantation should be kept free from all dead decaying matter which harbors saprophytes. The pails used for the collection of latex should be thoroughly scalded after using each day. Water used in cleaning the cups should be obtained from a source free from contamination. Collectors should never be allowed to obtain water for washing from contaminated streams. The factory and drying shed should be constructed according to the best accepted methods. The drying sheds should be located in a well-aërated place so as to provide for plenty of circulation, for rapid drying lessens the chances of spotting. Thin crêpe is less apt to become spotted, due to its quicker drying. Spotted rubber should never be packed with clean rubber. Usually these precautions are sufficient to prevent the trouble. In severe cases of infection it is advisable to sterilize the latex with 1 part of formalin to 400 parts of latex. Lightly spotted rubber may be somewhat cleared by rerolling the dried rubber and washing thoroughly with water.

OTHER FUNGI

A large number of apparently saprophytic organisms appear on the dead branches of Para rubber. Among these *Tryblidiella mindanaensis* Henn. and *Eutypella heveae* Yates have been identified.

HIBISCUS SABDARIFFA LINN. ROSELLE

BLIGHT: PHOMA SABDARIFFAE SACCARDO

Symptoms.—A stem blight that is rather severe on roselle, often killing entire plants. Diseased stems are attacked chiefly at the bases of small branches, at the nodes. Internodes also may be attacked. The spots spread until they entirely encircle the twigs. They are black with gray centers and are specked with minute black bodies. The disease is most severe on nearly matured plants.

Causal organism.—The minute black bodies are pycnidia. Upon crushing the pycnidia, a mass of small, one-celled, somewhat elongated, slightly olivaceous spores is expelled. The fungus grows well in pure culture, producing at first a growth of white mycelium, which later becomes studded with black pycnidia.

Control.—All diseased stems should be collected and burned. Crop rotation should be practiced.

IPOMOEA BATATAS POIR. SWEET POTATO

STORAGE ROT: LASIODIPLODIA THEOBROMAE (PAT.) GRIFFON ET MAUBLANC

Symptoms.—A common dry-storage rot, which is characterized by the production of a sooty mass of spores on the outside of infected potatoes. This disease is the same as that found upon the cacao fruit and other root crops and fruits (Plate XIX, fig. 5).

Causal organism.—The organism causing the disease is identical with that described under cacao. Cross inoculations from the fungus on cacao fruit to the sweet potato or vice versa can be easily carried out. The mycelium penetrates throughout the root and accumulates under the surface to produce a series of pycnidia, from which the mass of black spores arises (fig. 20). The organism is more fully discussed under cacao.

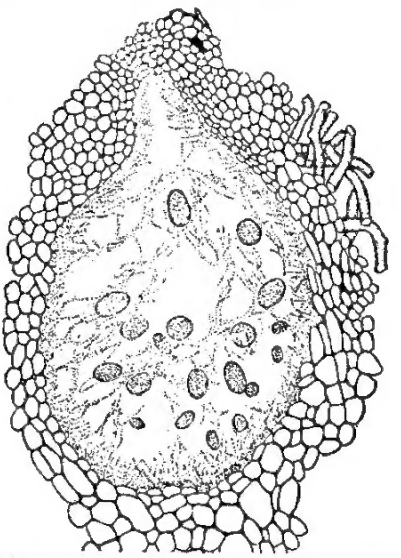

FIG. 20. *Lasiodiplodia theobromae* (Pat.) Griff. et Maubl. Section through diseased sweet potato, showing pycnidium, ostiolum, paraphyses, and immature spores (× 270).

Control.—Care should be used in digging the potatoes, so as to avoid injuries. The surface of the potatoes should be allowed to dry before storage. Storage should be in a well-ventilated place. All infected potatoes should be taken out and burned. Cacao fruits and root crops diseased with *Lasiodiplodia* must be kept away from stored sweet potatoes.

STORAGE ROT: RHIZOPUS

Symptoms.—A soft rot is frequently produced by this fungus. Diseased roots are soft and are covered with a black felty mold.

Causal organism.—This felty mass is made up of large num-

bers of sporangiophores and sporangia. The sporangia contain numerous black spores.

Control.—Sweet potatoes should be stored in a dry, well-aërated place. All rotted potatoes should be destroyed.

LACTUCA SATIVA LINN. LETTUCE

TIPBURN

Symptoms.—A nonparasitic disease that is common during the dry season. Leaves turn brown at the tip and gradually shrivel up.

(To be concluded.)

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